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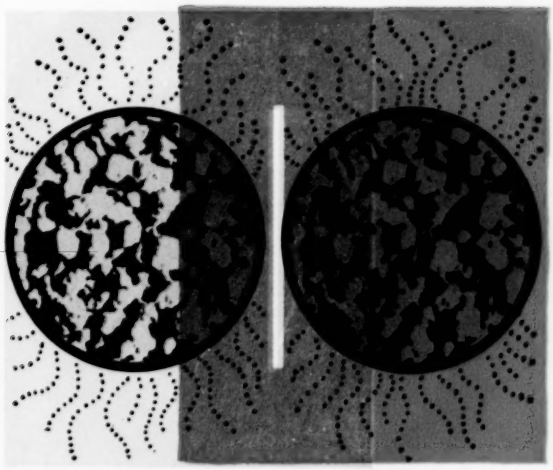

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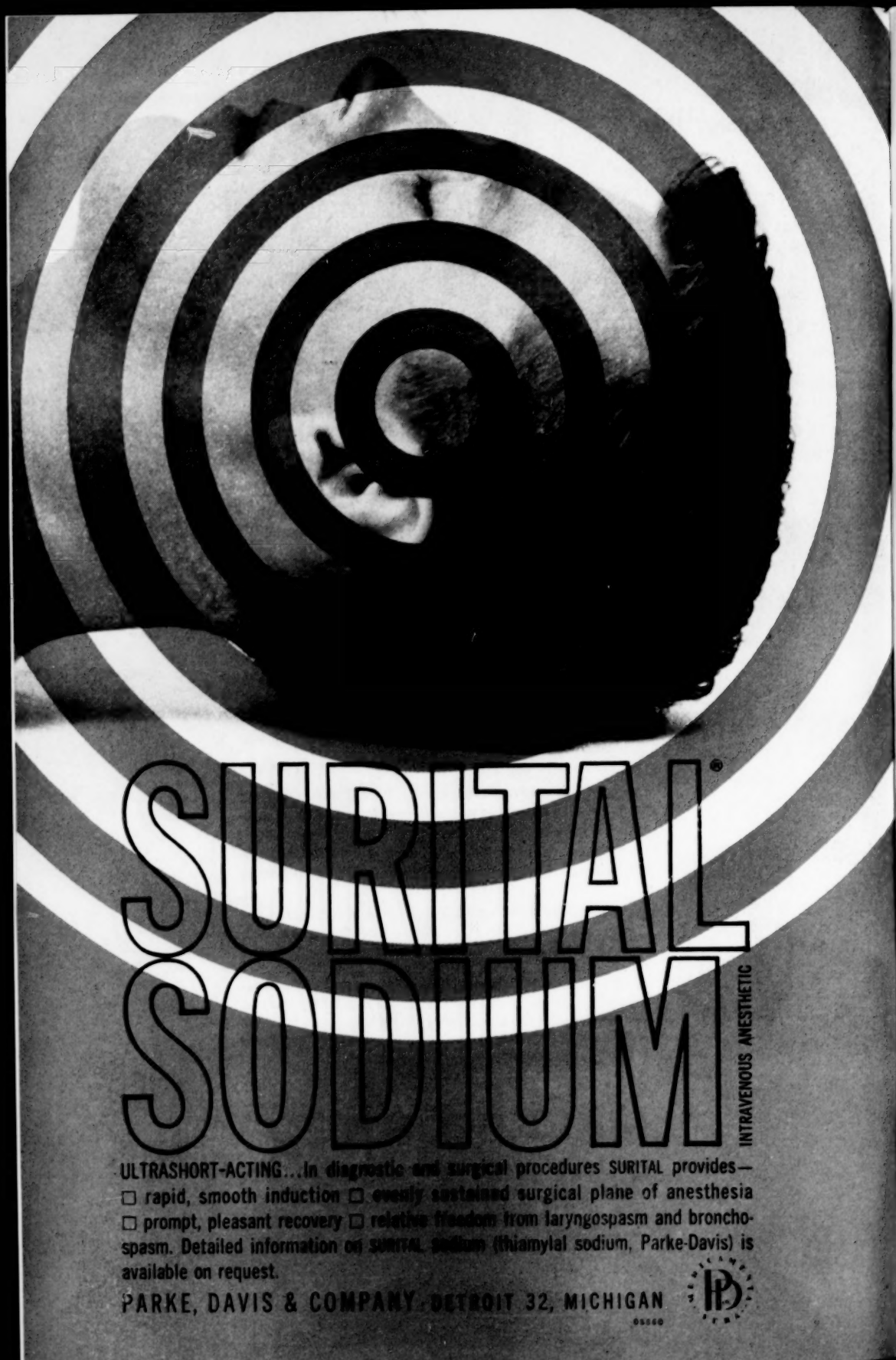

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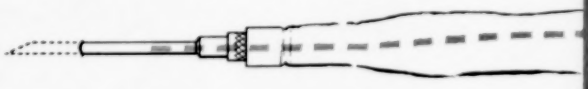

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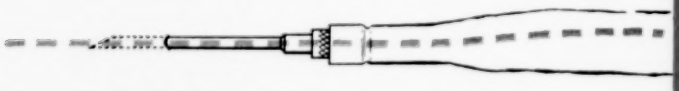

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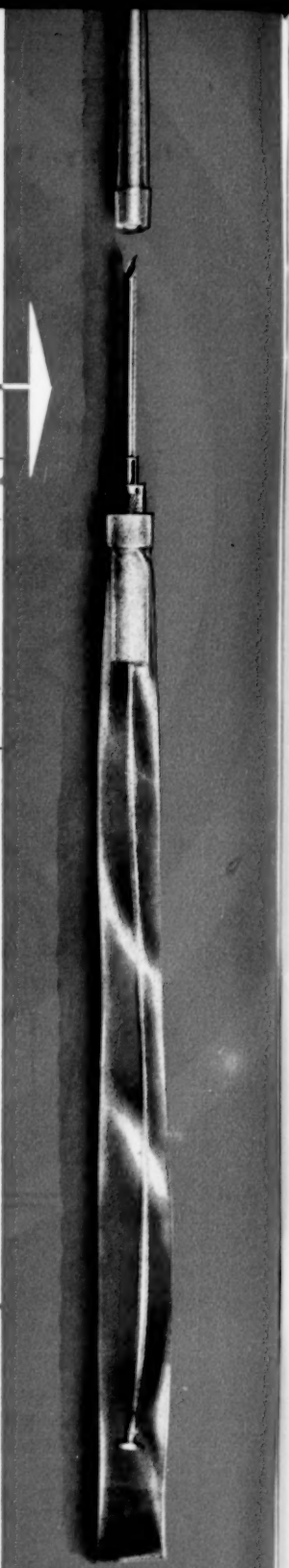

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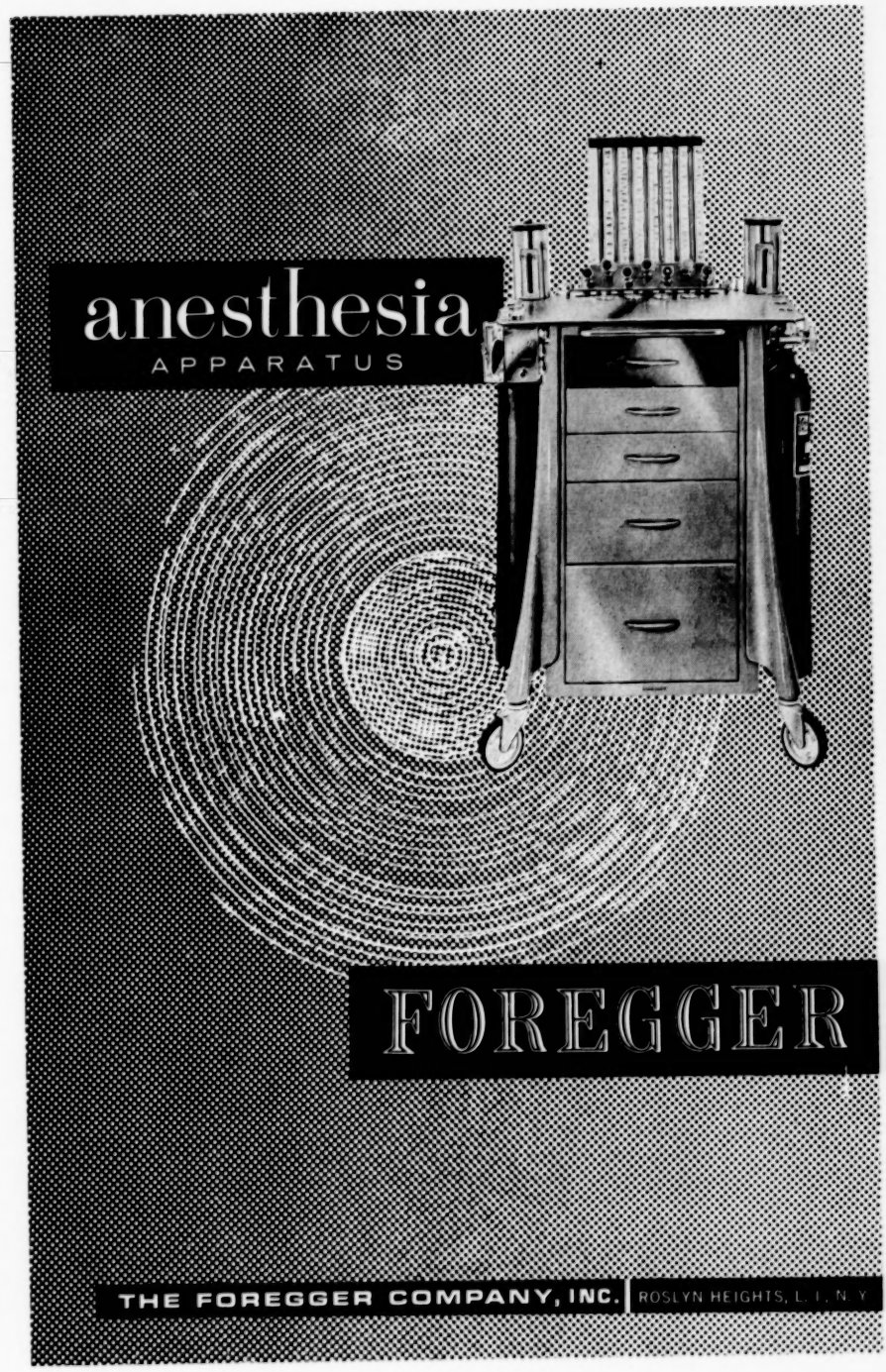
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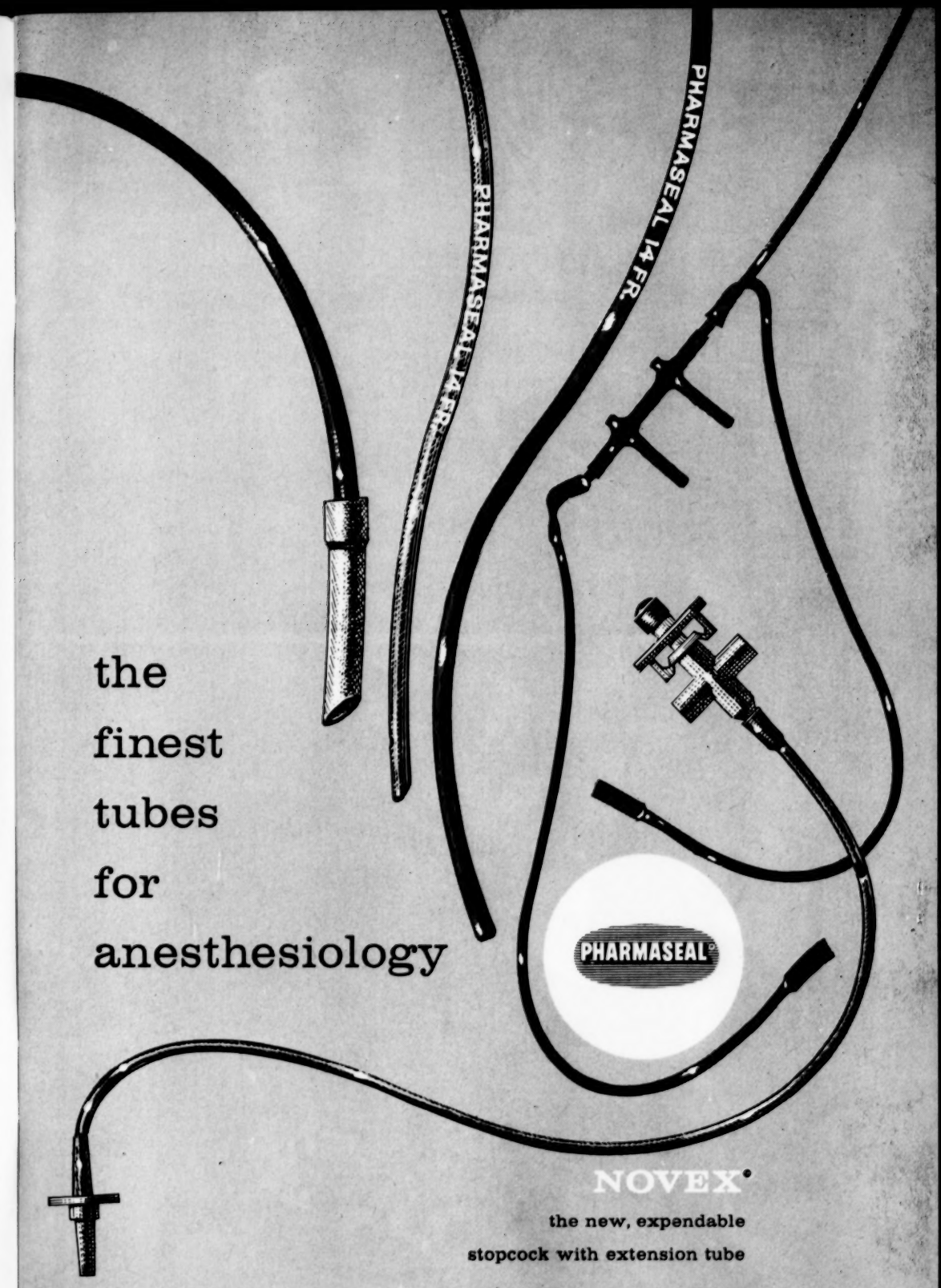

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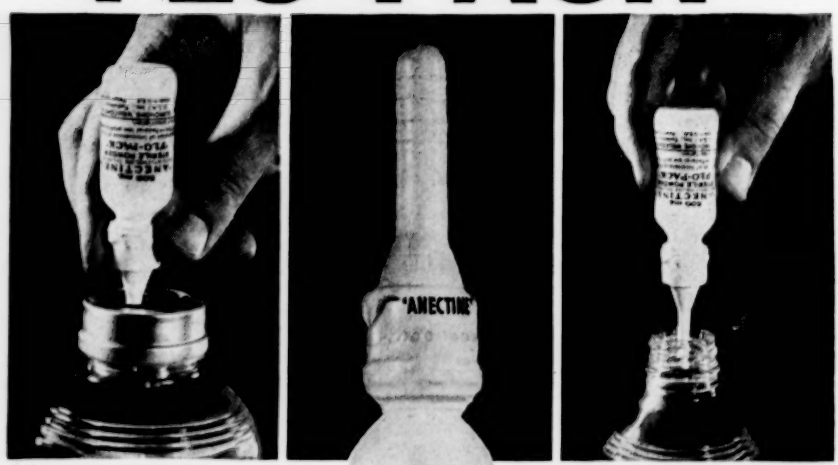

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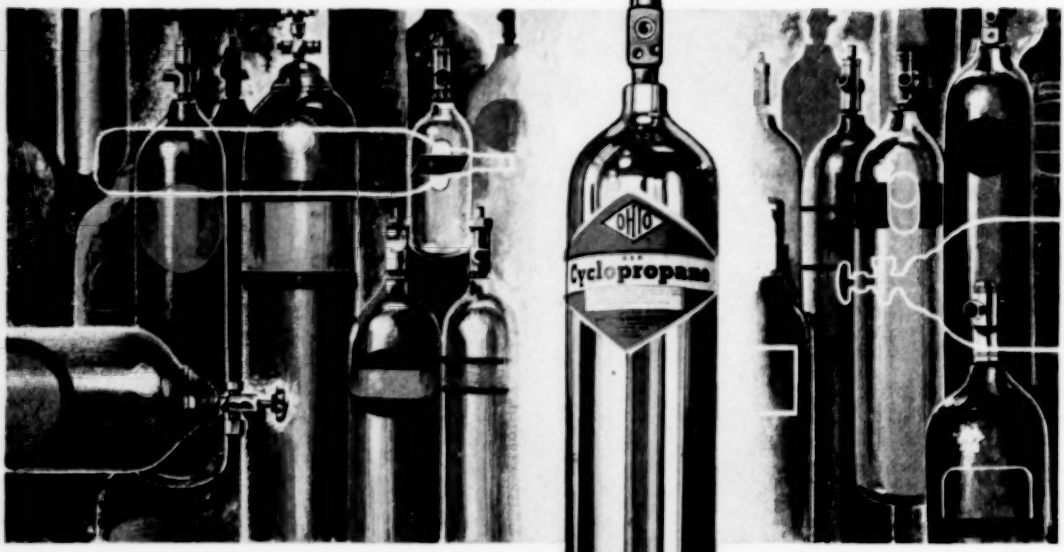

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Precision Balanced Anesthesia

John S. Lundy, M.D.*
Rochester, Minnesota

It is now almost 35 years ago that I introduced the technic of balanced anesthesia, and I gave the technic the name of "balanced anesthesia"[1] to denote the eminently useful effect obtained when preliminary medication, in the form of moderate doses of more than one drug, is administered. Anesthesia also was produced by the administration of small doses of several agents—whether local or general—so that it became possible to shorten the period required by the patient to recover from the effects of the drug compared to the recovery period which prevailed when the anesthetic agent consisted almost entirely of ether given by the semi-open drop method.

A few years later—1929—I began to administer sodium amytal intravenously; in 1931 I began to use pentobarbital sodium (Nembutal) intravenously; in 1932 I gave hexobarbital (Evipal) intravenously, and in 1934 I introduced the intravenous administration of thiopental sodium (Pentothal Sodium). Even after these advances I had no good reason to change the term, "balanced anesthesia." In 1942 solution of curare was employed to produce deep surgical relaxation and edrophonium chloride (Tensilon Chloride) later was used to terminate the effect of curare.

The great desideratum of adding precise control to balanced anesthesia was not yet achieved, but gradually the usefulness of the concept of antagonizing the physiologically depressing effect of an agent came to be realized. Over the last 50 or more years many workers in anesthesiology of long experience can recall experiments in which the physiologic processes in animals were greatly depressed by the effects of magnesium sulfate and were promptly relieved by the use of calcium chloride.[2] Sollmann[2] wrote of ionic antagonisms: "Calcium antagonizes most of the actions of magnesium. . . ." At that time we appreciated the spectacular effect produced by the antagonist, calcium chloride, but most observers regarded the phenomenon simply as an interesting demonstration which did not have any significant general application.

Today the factor of precision is readily available in anesthesiology, and so we have at last true "precision balanced anesthesia." Now, with such antagonists available as the analeptic, megimide (Bemegride),[3] and such stimulants of the central nervous system as mephentermine sulfate

* Section of Anesthesiology, Mayo Clinic and Mayo Foundation.

The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

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(Wyamine) and methyl-phenidylacetate hydrochloride (Ritalin Hydrochloride), and with the extremely useful antagonist, levallorphan tartrate (Lorfan Tartrate) it is possible more or less to terminate the effect of certain drugs which produce useful anesthesia and analgesia.[3] This is a rational extension of and a refinement of the "balanced anesthesia" of 1925.

Drugs and agents other than the ones I have mentioned can be used with success in ensuring precision balanced anesthesia. I might describe many of them, if limitation of space did not preclude such a description.

Let me refer to a rather typical adult patient who is to undergo an operation for mastoiditis. Precision balanced anesthesia requires that the patient be visited the night before the operation, at which time the patient is questioned about previous experience with anesthetic agents and whether or not an allergic sensitivity to some agent or preparation is present. The patient is then asked not to smoke until after the operation the next day. Abstinence from smoking affords the patient the best chance possible of avoiding coughing and the expectoration of material such as mucous plugs and the like from the lungs. Hence, cessation of smoking until after the operation is a very good precaution, and one which will make things easier for all concerned after anesthesia has been commenced or ended. Medication for the night prior to the operation is 25 mg. of promethazine hydrochloride (Phenergan Hydrochloride) and 500 mg. of ethchlorvynol (Placidyl) or glutethimide (Doriden), or methylprylon (Noludar) or ethinamate (Valmid) by mouth when it is time for the patient to go to sleep. This is done to produce deep and dreamless sleep. In most cases the patient will be reposing in a bed which is unfamiliar to him, and he needs to obtain as many hours of sound, deep sleep as possible so that he will have the strength and resistance required by the operation the next day. Orders are then written for medication for the next morning. This medication, for the average adult male person, consists of 2 mg. of levorphan tartrate (Levo-dromoran Tartrate), 1 mg. of levallorphan tartrate (Lorfan Tartrate) and 25 mg. of promethazine hydrochloride (Phenergan Hydrochloride).

As he comes to the operating room a half-hour or more after the preliminary medication for the operation has been given, the patient will be somewhat drowsy, but will answer questions well. A needle is placed in a vein, and to this needle a syringe and tubing for the administration of Pentothal Sodium are attached, with other tubing and other syringes, so that succinylcholine can be administered for intubation, and muscular relaxation can be maintained by the administration of curare.

Sometimes an intratracheal tube is to be used. When this is the case, the patient's arm (the one which is free) is flexed and he is asked to point his thumb at the ceiling as long as he can. Then the person who is to carry out intubation will announce how he plans to do it, not only in respect to the time factor but according to the dose of drugs. His assistant will inject 1 cc. of a 2.5 per cent solution of Pentothal Sodium every 10 seconds and announce the action by counting 1, 2, 3, 4 cc. and so on. The patient can hold his thumb up longer than he can count, and for this reason the

foregoing procedure is a very good way of individualizing the dose of Pentothal Sodium. In addition, there is a safeguard in the fact that if no results appear by the time 8 or 9 cc. has been injected, the needle unquestionably has been dislodged from the vein, because this dose is the average anesthetizing dose.

As soon as the patient's hand falls 1, 1.5 or 2 cc. of solution of succinylcholine is injected, the solution containing 20 mg. of the agent per cubic centimeter.

Now the patient's head is extended and the anesthetist's right thumb is placed on the patient's chin to depress the jaw and to open the patient's mouth. With the left hand adhesive tape is put on the upper lip to cover the upper teeth. The tip of the blade of the laryngoscope is inserted between the jaws as soon as fasciculations caused by the solution of succinylcholine have subsided and the jaw is relaxed. The left hand of the anesthetist steadies the laryngoscope and the right hand elevates the patient's jaw toward the ceiling and places the epiglottis on the end of the blade. This maneuver will avoid the inadvertent breaking of teeth and will ensure additional space in the throat for the tube.

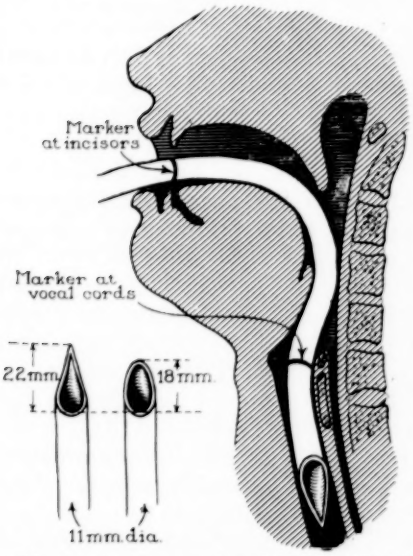

Fig. 1. A new pen-point tip for the Magill tube for endotracheal anesthesia, with markings at the level of the vocal cords and at the site of the upper central incisor teeth, to ensure that the point of the tube will remain approximately midway between the vocal cords and the bifurcation of the trachea.

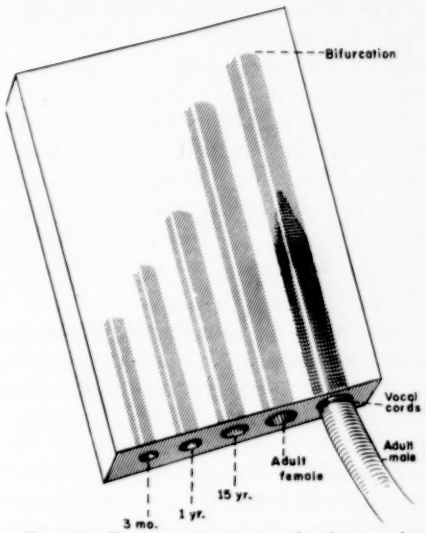

Fig. 2. Transparent gauge, the lower edge of which represents the level of the vocal cords. The tip of the hollow portion represents the bifurcation of the trachea. The tubelike holes have lengths graduated according to age and sex of possible patients. Tubes that are on hand can be marked with ink.

Next, the larynx is sprayed with a 10 per cent solution of cocaine and the intratracheal tube is inserted until the mark on the tube is still visible above the level of the vocal cords. A mark is then made on the tube at the point at which it touches the upper central incisor teeth (fig. 1). I have described a transparent gauge which is most useful in determining the level of the vocal cords in infants of 3 months, children a year old, persons 15 years old and adult persons (fig. 2).

The tube now constitutes an airway, through which the flow of gas usually will be 7 liters of nitrous oxide per minute and 3 liters of oxygen per minute. Intermittent doses of Pentothal Sodium and curare thenceforward keep the patient quiet. For some persons the preliminary medication will not be adequate, which means that some of it should be repeated.

Difficulty in anesthesia is recognized as imminent if, after as much as 0.5 gm. of Pentothal Sodium has been administered during the very first part of anesthesia, a good response is not obtained. If this unfavorable condition is allowed to proceed, it may be found that at the end of a 2-hour operation 2 gm. or more of Pentothal Sodium has been given. This is decidedly undesirable. It is far better to restrict the total dose of Pentothal Sodium to 1 gm. for an operation of 2 hours. Yet, when this sort of difficulty in anesthesia arises measures which will combat it can be brought into play quickly. The agent has the generic name, "phenazocine," and the trade name is "Prinadol."* It produces an analgesic effect. This drug can be administered intravenously. If, say, the patient is an adult male, the dose would be 5 mg. However, the administration of phenazocine will be followed in a matter of minutes (4 or less) by depressed respirations. But respiratory depression is promptly relieved by the intravenous injection of 1 mg. of Lorfan Tartrate.

In most cases, when these measures have been used, the operation may continue without the need for additional quantities of Pentothal Sodium or curare, and the patient will be well ventilated by use of a mixture of 30 per cent oxygen and 70 per cent nitrous oxide. It is most satisfying to any anesthetist to be able thus to obviate what otherwise would have been difficult and unsatisfactory anesthesia and relaxation without recourse to large quantities of drugs.

The factor of precision in balanced anesthesia also is employed even when the operation is concluded. At that time 5 mg. of Megimide is administered intravenously for each 100 mg. of Pentothal Sodium that has been used. After 3 or 4 minutes, if there is no satisfactory response from the patient in the matter of answering questions, even though respiration is improved and even if respiration is satisfactory, the dose of Megimide should be repeated. This action sharpens the patient's reflexes and brings him to the status in which he is able to respond to questions. To terminate the effect of the solution of curare, Tensilon Chloride is administered in a dose which depends upon the amount of solution of curare that has been used. Usually the dose will be 1 to 2 cc. of Tensilon Chloride. After this has been given, improvement in respiration may be observed again. As I have said, the effect of Prinadol is analgesia, but it also causes respiratory depression. Still, by resort to the carefully calculated dose of Lorfan Tartrate, respiratory depression can be eliminated and analgesia can be retained. This is another instance of the information which must be readily at hand if the factor of precision is to be maintained in balanced anesthesia. The mode of action is not drug against drug, but rather, dose of drug against dose of drug. A practical example will illustrate the principles involved.

* Smith, Kline & French Laboratories.

To three rabbits we administered 0.4 mg. of Prinadol per kilogram of body weight. Immediately the eyes closed, the ears drooped, the back sank a little and respirations became slow. Any change in the respiration of the rabbit is very noticeable, since this animal breathes rapidly. To one of these rabbits which had received Prinadol we gave a total dose of 1 mg. of Lorfan Tartrate intravenously. Within 15 seconds the animal opened the eyes; the ears stood up and the rabbit walked around breathing normally. The analgesia had disappeared. To another rabbit we administered a dose of Lorfan Tartrate reduced to 0.5 mg.; within 15 seconds the animal opened the eyes, the ears stood up and the animal was able to stand up. There was no respiratory depression but the analgesia that was produced would have been of little use. To the third rabbit I administered 0.25 mg. of Lorfan Tartrate intravenously. Within 15 seconds the animal opened the eyes, the ears stood up, respirations were normal and it was very difficult to produce any indication of pain by pinching the ears, stepping on the tail and so on.

Now, the latter condition is the useful state of analgesia which I wish to produce, and the proportion of 5 to 1 seems to be about the same for patients and for rabbits. In human beings the dose would be 5 mg. of Prinadol against 1 mg. of Lorfan Tartrate. Fortunately, Prinadol and Lorfan Tartrate can be taken by mouth.

I believe that with more experience I may be able to introduce the use of these drugs into ordinary dental practice, with the objective of relieving such patients of their fear of the dentist and at the same time ensuring safe relief of pain in the dental office. Many oral surgeons administer Pentothal Sodium intravenously in office practice and they are, of course, unable to avoid occasional laryngospasms. Sometimes the situation can become unsafe. I am hoping that as I continue to use Prinadol the laryngospasm that has been absent thus far will not appear, so that with the administration of 50 per cent nitrous oxide and 50 per cent oxygen, patients will be able to open and close their eyes as requested and can undergo an operation without pain and in perfect safety. A local anesthetic agent may be substituted for the nitrous oxide and oxygen, and thus far this has been done with satisfactory results.

Prinadol is prepared especially for patients who have chronic arthritis with pain. The drug has at least five times the analgesic power of morphine, yet the factor of addiction is low, possibly, we hope, not greater than that of codeine or at least morphine. Prinadol occupies a very, very useful place in precision balanced anesthesia, and I feel fortunate to have seen this great advancement take place in our ability to control anesthesia for patients.

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[1] Lundy, J. S.: Balanced Anesthesia. Minnesota Med. 9:399-404 (July) 1926.

[2] Sollmann, T. H.: A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology. Philadelphia, W. B. Saunders Company, 8th ed., p. 1062, 1957.

[3] New and Nonofficial Drugs. J.A.M.A. *170*:1316-1317 (July 11) 1959.

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Respiratory Physiology and Air Travel

Esther M. Greisheimer, M.D., Ph.D.*
Philadelphia, Pennsylvania

Definition of Respiration

Respiration is the process of supplying the body with oxygen and removing the carbon dioxide. It is divided into external and internal phases. External respiration is concerned with getting oxygen from the environment into the blood and removing carbon dioxide from the blood to the environment. Internal respiration deals with transportation of oxygen from the lung to the tissues and the chemical reactions in the cells by which energy is released from foodstuffs and carbon dioxide is transported from the tissues to the lungs. Cellular respiration will not be included in this review.

Movements of Respiration

In order to move air into the lungs a passageway is needed, and a bellows arrangement. The thoracic cage serves as the bellows and it is operated by skeletal muscles, which are controlled by somatic nerves. The thoracic cavity is enlarged by contraction of the diaphragm and external intercostal muscles and, as the pressure inside the lungs is lowered, air is forced in from the atmosphere. This process is called inspiration. When the diaphragm and external intercostal muscles relax, the air inside the lungs is put under greater pressure, due to decrease in size of the thorax, and will be forced out until the pressure inside is again equal to atmospheric. This process is called expiration.

The lungs make available an enormous moist surface for gaseous exchanges. The alveolar surface is from forty to fifty times as great as the body surface. In the lungs there are only two thin membranes between the capillary blood and the alveolar air. The respiratory gases diffuse readily through these moist membranes due to differences in partial pressure of the gases on the two sides of the membranes. Molecules of gases diffuse from the higher to the lower pressure in each case.

Pressure Relationships

A. Intrapulmonic pressure. Intrapulmonic pressure refers to the pressure inside the lungs. It is constantly changing. During inspiration it is lower than atmospheric, consequently air enters the lungs until intrapulmonic and atmospheric pressures are equal, which occurs at the end of inspiration, and air flow ceases. During expiration the intrapulmonic pressure is above atmospheric, due to decrease in size of the thorax, consequently air flows out until intrapul-

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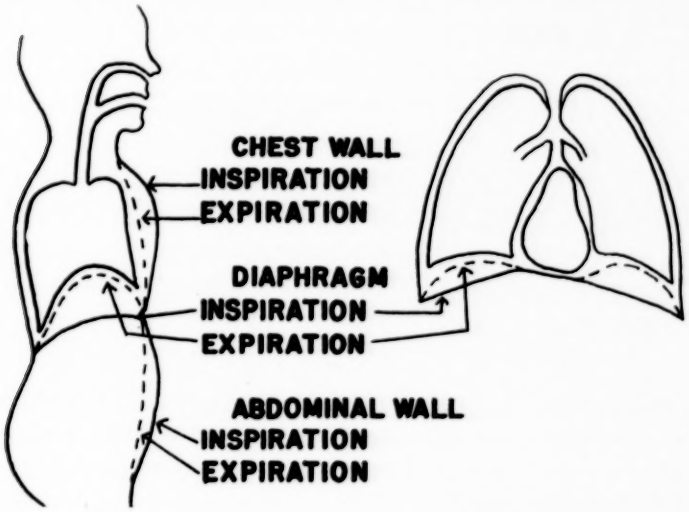
monic pressure is again equal to atmospheric pressure, and air flow ceases.

When you assist or control respiration manually, you force air intermittently into the lungs by creating a pressure above atmospheric pressure outside. You expand the thorax by force, with or without muscular effort on the part of the patient. When you release the pressure the thorax decreases in size and air flows out. Controlled respiration differs from spontaneous respiration in that the intrapulmonic pressure is never lower than atmospheric pressure.

Mechanical devices for administering artificial respiration also force air into the lungs by positive pressure outside, without the benefit of negative pressure inside the lungs. Sometimes expiration is assisted by the application of negative pressure outside. The physiological aspects of artificial ventilation have been ably discussed by Nunn[1].

B. Intrathoracic or intrapleural pressure. Intrathoracic or intrapleural pressure refers to the pressure between the lungs and thoracic wall, or between the two layers of pleura. This pressure is normally negative during the entire respiratory cycle when one breathes spontaneously. It is more negative during inspiration, due to the greater elastic recoil of the



	INTRAPULMONIC (mm.Hg)	INTRAPLEURAL (mm.Hg)
NORMAL INSPIRATION	−2 TO −3	−5 TO −10
NORMAL EXPIRATION	+2 TO +3	−3 TO −5
FORCED INSPIRATION		−30 TO −80
FORCED EXPIRATION		+100 TO +150

Figure 1. Changes in position of chest wall, abdominal wall and diaphragm in inspiration and expiration. Changes in intrapulmonic and intrapleural pressures during normal respiration, and in intrapleural pressure during forced respiratory movements.

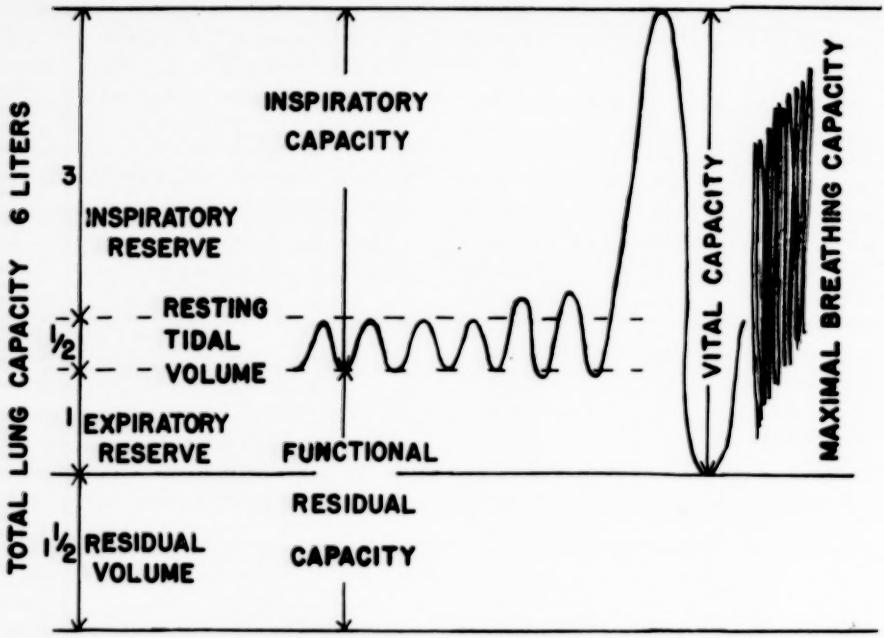


Figure 2. Various lung volumes and capacities, drawn to scale.

distended lungs. The changes in intrapulmonic and intrathoracic pressure during inspiration and expiration are shown in Fig. 1.

The intrathoracic pressure becomes positive when one performs the "Valsalva" maneuver, such as straining at stool. This is a forced expiration with the glottis closed. The change from negative to a markedly positive intrathoracic pressure has striking effects in that it alters venous return, cardiac output and arterial blood pressure. Straining at stool is an especially dangerous act for a patient with a damaged heart muscle or sclerotic blood vessels, and this is the reason many cardiac deaths occur in the bathroom.

Pulmonary Function Tests

A. Lung volumes. The inspiratory reserve volume is determined by having the patient take as deep a breath as possible, beyond the usual tidal amount. It is normally between 2½ and 3 liters. The inspiratory reserve plus the tidal volume is a measure of the inspiratory capacity.

The expiratory reserve volume is the amount of air an individual can exhale, beyond the tidal volume, by making a forced expiration, following a normal inspiration. It is about one liter.

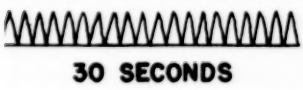
The vital capacity is the sum of the tidal volume, inspiratory reserve volume and expiratory reserve volume. It is the volume of air one can exhale by the deepest possible expiration following the deepest possible inspiration. The usual value is between 4 and 4½ liters.

The residual volume is the amount of air which one cannot exhale even by the most powerful effort. It is forced out only by collapse of the lungs, as in pneumothorax. It is about

1½ liters. It varies with pulmonary disease, and is especially large in emphysema.

The functional residual capacity is the sum of the residual volume and the expiratory reserve volume. It is about 2½ liters.

The total lung capacity is about 6 liters. Figure 2 illustrates the total capacity of the lungs, and the volumes and capacities defined above. The ratio of the residual volume to the total lung capacity is between 20 and 35% in normal individuals. If the residual volume is increased, as it is in asthma or emphysema, the ratio is above the normal limit. If the total lung capacity is decreased, as it is in pulmonary fibrosis and pulmonary congestion, the ratio is likewise above the normal limit.



RATE 40; TIDAL VOLUME 200; MVR=8000cc.
DEAD SPACE 150cc.; 200-150=50X40=2000cc.

RATE 20; TIDAL VOLUME 400; MVR=8000cc.
DEAD SPACE 150cc.; 400-150=250X20=5000cc.

RATE 10; TIDAL VOLUME 800; MVR= 8000cc.
DEAD SPACE 150cc.; 800-150=650X10=6500cc.

Figure 3. Diagram illustrating effective pulmonary ventilation accomplished by slow, deep breathing in contrast to that of rapid, shallow respiration, despite identical minute volumes.

B. Pulmonary ventilation. (1) Volume of alveolar ventilation. The frequency of respiration can be counted by watching the patient's respiratory movements, or by recording them.

The tidal volume of respiration is the amount of air moving in and out during normal, quiet breathing. An average figure is ½ liter.

When the frequency and tidal volume are known, the minute volume can be calculated, as it is the product of the frequency and the tidal volume. It is normally between 7 and 8 liters.

The anatomical dead space is the volume of the passageway from the nose and mouth to the alveoli. The air in the dead space plays no part in the gaseous exchanges. It amounts to about 150 cc. in men and 110 cc. in women. It varies with endotracheal tubes, masks and location of valves, in anesthesia. It has been shown recently that the position of the jaw exerts an important influence on the volume of the anatomical dead space[2].

The physiological dead space and the anatomical dead space are practically the same in healthy individuals, but they differ in patients who have pulmonary disease. In such cases, the physiological dead space includes two volumes in addition to the anatomical dead space volume.

These are (1) the volume of inspired gas that is distributed to alveoli which have no capillary blood flow, and (2) the volume of inspired gas that hyperventilates some alveoli. In either of these conditions the efficiency of ventilation is lowered.

Alveolar ventilation per minute may be calculated by subtracting the dead space volume from the tidal volume, and multiplying the difference by the frequency. Figure 3 illustrates the inefficient ventilation accomplished by rapid, shallow respiration in contrast to the efficient ventilation accomplished by slow, deep respiration.

It is of interest to consider the per cent renewal of the alveolar air. At the end of a normal expiration, the lungs contain the functional residual air (2½ liters) and the passages also contain alveolar air (0.150 liter). With a tidal volume of 0.5 liter, only 0.350 liter mixes with the alveolar air and 0.150 liter remains in the passages. The actual per cent renewal is small (0.35 tidal-2.65), being about 13%.

(2) Uniformity of alveolar ventilation. The ideal ventilation for any individual is that which enables him to maintain the partial pressures of oxygen and carbon dioxide in the alveolar air at 100 mm. Hg and 40 mm. Hg, respectively. The ventilation should supply as much oxygen to the alveoli in a unit time as is removed by the pulmonary capillary blood.

Uneven ventilation may occur in the lungs. There may be changes in the elasticity of some areas which will interfere with ventilation. In some individuals there are some bronchi which are partially obstructed. Congestion interferes with expansion of alveoli. The blood flow may vary in different areas. So far as anesthesia is concerned, the opening of the thorax alters ventilation, and certain surgical positions interfere with ventilation. The end result of uneven ventilation, whatever the cause, is hypoxia.

The time allotted to pulmonary function tests is short, although this section of physiology of respiration is receiving such widespread attention in clinics at present. I shall suggest sources for further information. Campbell[3] has published a guide to the terminology and symbols used in respiratory physiology, which is of value to anesthetists. Comroe et al[4] give detailed information on the pulmonary function tests, and Fowler and Miller[5] have published a paper of value on the physiologic considerations in the diagnosis and treatment of ventilatory insufficiency.

Mechanics of Breathing

Comroe et al[4] give an excellent discussion of the mechanics of breathing, and a review of their work will be presented.

A. Principles of the mechanics of breathing. (1) Mechanical factors involved in inspiration. There are some elastic tissues in the lungs and thorax and these must be stretched during inspiration. The force to stretch them is supplied by the contraction of the muscles of inspiration. When the muscles relax the elastic tissues return to the resting position. The force is measured as the pressure necessary to stretch the elastic tissues. The stretch is measured as the volume of air or oxygen used to distend the lungs. The pressure and volume are measured in the static condition, that is, when no air is flowing. When pressure and volume are plotted against each other the distensibility of lungs and thorax or

stiffness is indicated. The relation between pressure and volume is called "compliance". If there is congestion or fibrosis of the lung tissue, the increase in volume for a given amount of pressure will be less than in the normal lung, and this is called a decrease in compliance.

There are tissues in the thorax which are nonelastic, and a certain amount of force is required to overcome the friction within these tissues when they are moved during inspiration. The amount of force required depends on the speed with which the tissues are moved.

There is a third element of force required during inspiration. This is the force needed to overcome the resistance offered to the flow of air through the tracheobronchial tree. There is friction within the air stream itself, and between it and the walls of the respiratory passages. This force is the pressure difference between the atmosphere and the alveoli. The amount of pressure required depends on the speed of air flow and the resistance to the flow. The airway resistance depends on the number, length and diameter of the passages into the lungs. The smaller the diameter, the greater the resistance. The use of an endotracheal tube which is long and narrow increases the resistance to air flow.

(2) Mechanical factors involved in expiration. When the muscles of inspiration contract part of the energy produced is stored as potential energy in the elastic tissues of the lungs and thorax. When the muscles relax, the stretched elastic tissues return within 3 seconds to their resting length, in the normal individual.

Some force is required to overcome the nonelastic tissue resistance, or the friction of motion.

The airway resistance is no greater in expiration than in inspiration in the normal individual. In such conditions as asthma and emphysema the airway resistance is much greater during expiration than during inspiration. This means that the expiratory phase is not completed within 3 seconds, and the functional residual capacity is greater than normal.

B. Tests used in determining the mechanics of breathing. 1. Compliance of the lungs and thorax may be measured in various ways. One of the simpler methods is to inspire a measured volume of air or oxygen from the spirometer, then close the nose and mouth, open the glottis and relax the muscles of respiration. The alveolar pressure can be measured by nasal tube, when no air is flowing (static). Compliance is expressed as liters per cm. of water pressure. Comroe et al[4] give the compliance of lungs and thorax as 0.13 liter per cm. of water pressure. The compliance values vary with different methods and authors.

2. Compliance of the lungs alone may be determined by measuring the static "transpulmonary pressure", which is the pressure difference between the pleural space and mouth. The intrapleural pressure can be measured fairly satisfactorily by means of a balloon in the lower third of the esophagus. When measurements are made at the end of inspiration, the compliance of the lungs alone averages 0.22 liter per cm. of water pressure. In other words, a given distention is accomplished with less pressure than when both lungs and thorax are considered as a unit. Posture influences the compliance of the lungs[6, 7]. Compliance is decreased in pulmonary congestion, fibrosis, asthma and in the supine position.

3. Mechanical resistance is determined by measuring the pressure necessary to produce a given air flow. It is expressed as cm. of water pressure per liter of air flow per second. Attinger[8] has measured the mechanical resistance (resistance to air flow per second and nonelastic resistance) in various patients with cardiopulmonary disease. It is high, as would be expected, in both asthma and emphysema. Bartlett et al[9] have described a method for measurement of airway resistance during any breathing pattern in man.

4. Ratio of inspiration to expiration. Normally expiration is slightly longer than inspiration (1.2:1). In asthma expiration may be 2 to 3 times as long as inspiration. Expiration is prolonged in emphysema, also. It has been stated that if the airway resistance is increased the time to return to the resting expiratory level is longer than the 3 seconds required in the normal.

5. Timed vital capacity. Gaensler[10, 11] was one of the first to call attention to the fact that the results of a vital capacity test can be misleading, as there is no consideration of time. He determined the per cent of the total vital capacity which was exhaled in 1, 2 and 3 seconds. More recently, it has been suggested that the per cents for 0.5, 0.75 and 1 second should be determined[12]. The individual makes a maximal inspiratory effort and then without hesitation makes a full expiration, with maximal force. The amounts exhaled at the above times are noted. Since several groups of investigators have determined the values for one second, 2 seconds and 3 seconds, only these values will be given. A normal individual should exhale about 83% within 1 second, 94% within 2 seconds and 97% of the total vital capacity within 3 seconds. The lowest limit of normal is 75% of the total exhaled in 1 second.

The timed vital capacity is a satisfactory way of separating obstructive from non-obstructive ventilatory defects. The former type of defect is characterized by a low value or slow rate of exhalation, due to increased airway resistance (asthma and emphysema).

6. Maximal breathing capacity. This refers to the maximal amount of air one can move in and out of the lungs by breathing as rapidly and as deeply as possible for 15 seconds. It is calculated in terms of one minute, and is about 100 liters per minute for a normal adult man. It is not too dependable as a test, since it is affected by muscular weakness and by the resistance to air flow in the respiratory passages. It requires the full cooperation of the patient.

Figure 4 is a table of comparative values of the results of some pulmonary function tests and tests of the mechanics of breathing in the normal, asthmatic and emphysematous individual.

Control of Respiration

A. Nervous control. The concept of the nervous control of respiration has been revised within the last few years[13]. There is thought to be a "postural" element in respiration, which is based on tonic innervation of both inspiratory and expiratory muscles, just as one finds in the flexors and extensors of the extremities. The postural tone is influenced by impulses from the cerebral reticular formation, shown in Fig. 5. The phasic movements of respiration (rhythmic inspiration and expiration)

are superimposed on the postural muscle tone.

It is thought that there is a respiratory center in the medulla, which is capable of maintaining rhythmic respiration, even when the parts of the brain above this level have been severed. Under these conditions the individual breaths vary in rate and amplitude, giving rise to the descriptive term "ataxic" eupnea. The medullary center is capable of another type of activity, as shown by an occasional maximal respiratory effort, called an "all or nothing" breath. The thorax is held in the expiratory position. If the vagus nerves are cut in such an animal, very little change is noted in respiration. This seems to be adequate proof that there is no breath by breath control of respiration by impulses from the lungs by way of the vagus nerves. In other words, the "self-steering" mechanism

COMPARISON OF NORMAL, ASTHMATIC AND EMPHYSEMATOUS INDIVIDUALS

	NORMAL	ASTHMA	EMPHYSEMA
TIDAL VOLUME (ml)	500	600	330
RATE (PER MINUTE)	15	16	15
MINUTE VOLUME OF RESPIRATION (LITERS)	7·50	9·60	4·95
INSPIRATORY RESERVE (LITERS)	2·21	2·03	1·02
EXPIRATORY RESERVE (LITERS)	1·39	0·92	0·50
VITAL CAPACITY (LITERS)	4·10	2·80	1·73
TIMED VITAL CAPACITY – % OF TOTAL EXHALED IN 1 MINUTE	83	43	47
MAXIMAL BREATHING CAPACITY (LITERS/MINUTE)	100	62	56
RESIDUAL VOLUME (LITERS)	1·69	3·50	4·40
FUNCTIONAL RESIDUAL CAPACITY (LITERS)	3·08	4·42	4·90
TOTAL LUNG CAPACITY (LITERS)	5·79	7·05	6·25
ALVEOLAR VENTILATION (ml)	5250	7200	2700
AIRWAY RESISTANCE (cm H_2O PRESSURE PER LITER OF AIR FLOW PER SECOND)			
INSPIRATION	2·95	11·90	9·73
EXPIRATION	3·45	16·86	17·54
% O_2 SATURATION IN ARTERIAL BLOOD	97	90	90
CO_2 TENSION IN ARTERIAL BLOOD (mm. Hg)	40	38	57
pH – ARTERIAL BLOOD	7·40	7·44	7·33

Figure 4. Table of comparative values in normal, asthmatic and emphysematous individuals, compiled from published results of numerous authors. The timed vital capacity is the amount of the total exhaled in one second (not minute). Airway resistance should read mechanical resistance, since it includes resistance to tissue deformation or that due to tissue viscosity, as well as that due to air flow per se.

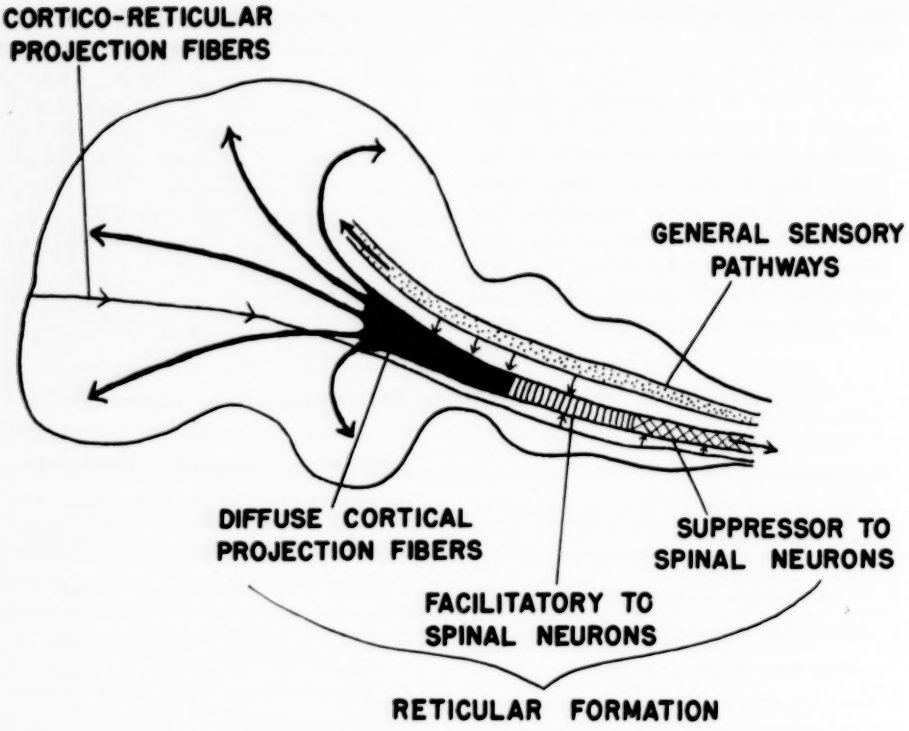


Figure 5. A diagram of the cerebral reticular formation. Collaterals to reticular formation from general sensory pathways are shown. The ascending fibers from the reticular formation to the cerebral cortex, which give rise to the "alerting" response are shown, as well as the facilitatory and suppressor areas which exert important effects on activities mediated by the spinal cord.

or Hering-Breuer reflexes are under question with respect to continuous respiratory control.

The area of the brainstem formerly designated the apneustic center is apparently only a part of the facilitatory portion of the reticular formation[14]. When this region and parts below are intact, and all parts above destroyed, the all or nothing breaths appear more often than in the medullary preparation. There is an increase in the inspiratory tone of the thorax, which now occupies a mid-position of inspiration.

If the vagus nerves are cut in such a preparation "apneustic" breathing appears due to unopposed facilitation by way of the reticular system. It may be considered a manifestation of decerebrate rigidity of the respiratory system. There are inspiratory spasms, due to increase in tonus of the muscles of neck, thorax and abdomen. The inspiratory spasms may last from 15 seconds to 4 minutes each. If one observes closely, he will see minute rhythmic respiratory movements superimposed on each inspiratory spasm. These have a small amplitude, and constitute a sort of fringe on top of the inspiratory spasm. Such respiration may continue for several hours. This seems to indicate that the facilitatory area of the reticular formation affects the muscles of respiration as well as the somatic muscles of the extremities, abdomen and neck, to-

gether with the smooth muscles of various bodily structures, in decerebrate rigidity. Fig. 6 illustrates the types of respiration seen in animals when transections are made at various levels of the central nervous system.

The area in the pons formerly called the pneumotaxic center is apparently a part of the suppressor area of the reticular formation. Respiratory muscles, as well as other somatic muscles, and smooth muscles, are affected by impulses from the suppressor area of the reticular formation.

It is probable that the vagus nerves do not regulate respiration, breath by breath, but influence it only because they carry afferent impulses to the suppressor area of the reticular formation from various receptors of the body, including the lungs. By this means, the vagus nerves exert only an important over-all inhibitory effect on respiration.

B. Chemical control of respiration. The activity of the respiratory center in the medulla is subject to chemical control by way of the carbon dioxide tension of arterial blood. Normally the center is very sensitive to the carbon dioxide tension, but in surgery it is depressed by preanesthetic medication and anesthetic agents. We might think of the chemical control as the fundamental influence which keeps us breathing, while the nerve impulses serve to regulate the activity of the center in accordance with changing requirements.

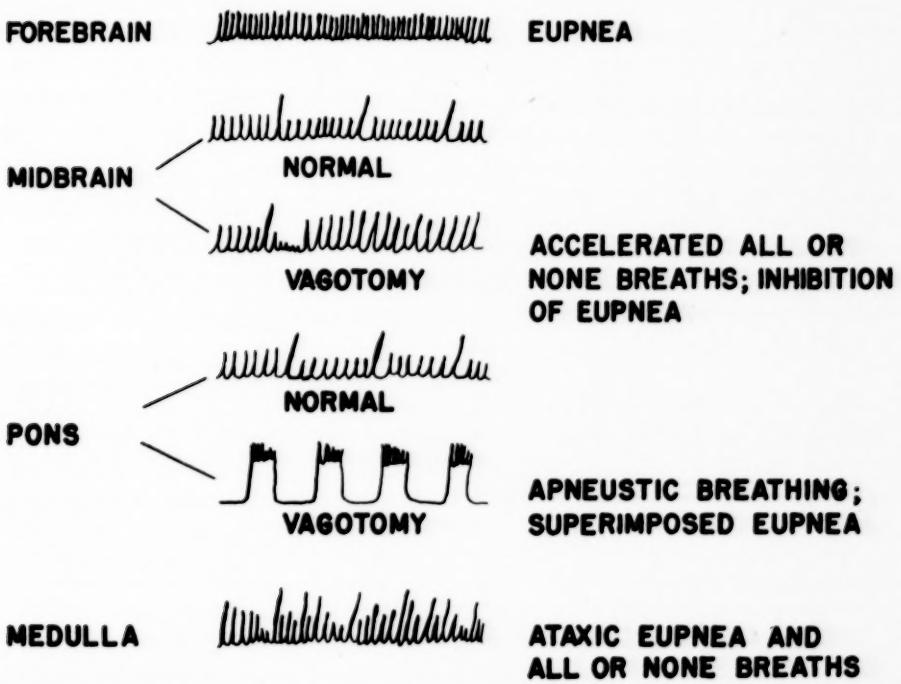


Figure 6. A diagram illustrating the types of breathing seen in animals whose brains are transected at various levels. The isolated medulla is capable of rhythmic respiratory movements, which vary in amplitude. The addition of the pons alone or both pons and midbrain, makes the respiratory movements more regular and increases the all or nothing breaths. When the vagus nerves are cut in an animal with pons and medulla, apneustic breathing occurs. When the midbrain is present also, no apneusis is seen.

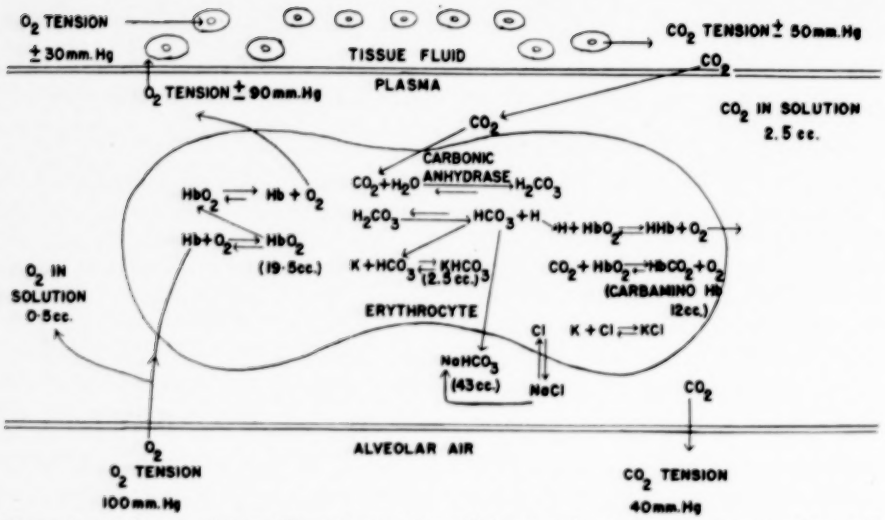


Figure 7. A diagram showing the main reactions which occur in erythrocytes in connection with transport of oxygen and carbon dioxide. At the left, the entrance of oxygen from alveolar air into pulmonary capillaries is shown below, and the exit of oxygen from the systemic capillaries into the tissue fluid and cells, above. At the right, the entrance of carbon dioxide from the cells and tissue fluid into systemic capillaries is shown above, and its eventual exit from the pulmonary capillaries into alveolar air, below.

Chemistry of Respiration

A. Composition of air. Inspired air contains 20.96% of oxygen and 0.04% of carbon dioxide; the remainder is nitrogen and rare gases. The water vapor varies, as does the temperature.

Alveolar air contains 14% of oxygen and 5.5% of carbon dioxide; it is saturated with water vapor and warmed to body temperature.

Expired air varies in composition, being made up of dead space air and alveolar air.

The tension of oxygen in inspired (dry) air is about 150 mm. Hg. In alveolar air it is about 100. The tension of carbon dioxide in alveolar air is about 40 mm. Hg.

B. Diffusion. The process of diffusion is responsible for the passage of oxygen from alveolar air to venous blood, in which the tension is lower. It is likewise responsible for the passage of carbon dioxide from the venous blood to alveolar air, in which the tension is lower. In the systemic capillaries, the oxygen diffuses from blood to tissue fluid and on to cells, and carbon dioxide diffuses from cells through tissue fluid and into blood. The gas molecules always diffuse from a higher to a lower pressure.

C. Transport of oxygen. Oxygen first dissolves in the plasma, then it diffuses into the red blood corpuscles, where it is combined with hemoglobin, as shown in Figure 7. The taking up of oxygen by hemoglobin is favored by the diminishing carbon dioxide tension and slightly increased alkalinity of the blood in the pulmonary capillaries. The combination of oxygen with hemoglobin is "loose", and when the blood reaches the systemic capillaries, the dissolved oxygen diffuses out into the tissue fluid and on to the tissue cells. At the same time, carbon dioxide is diffusing into the blood, and this favors the dissoci-

ation of oxyhemoglobin. As the carbon dioxide diffuses in and the oxyhemoglobin gives up its oxygen the blood becomes slightly less alkaline, and this favors further dissociation. The importance of carbon dioxide in the dissociation of oxyhemoglobin will be mentioned again when hyperventilation is considered. As oxygen is given up by the hemoglobin it leaves the erythrocytes and dissolves in the plasma, and then diffuses out to areas of lower oxygen tension.

D. Transport of carbon dioxide. The transport of carbon dioxide is complicated. As it diffuses into the blood from the tissue fluid, it dissolves in the plasma. About 2.5 cc. per 100 cc. of plasma are carried in solution. Most of it enters the erythrocytes, in which, under the influence of carbonic anhydrase, it reacts with water to form carbonic acid. This dissociates immediately, to form the carbonate radical (HCO_3) and H; the carbonate radical diffuses across the erythrocyte membrane into the plasma and unites with sodium to form sodium bicarbonate. About 43 cc. per 100 cc. of plasma are carried as sodium bicarbonate. The Cl which has been displaced from the Na in plasma enters the erythrocyte and unites with base. About 12 cc. of carbon dioxide are carried as carbamino compound in the red corpuscles, as a result of interaction with amino acids of hemoglobin. Some carbon dioxide unites with potassium to form $KHCO_3$, in red corpuscles; in this way about 2.5 cc. more of carbon dioxide are carried. Figure 7 shows by diagram the various ways in which carbon dioxide is transported.

In the lungs the processes which occurred in the systemic capillaries are reversed, carbon dioxide is released and the chloride returns to the plasma to form NaCl again. The moving in and out of erythrocytes is called the chloride shift.

E. Transport of nitrogen. Nitrogen, which composes about 79% of inspired air, is carried only in solution in the plasma. It diffuses from the plasma throughout the fluids of the body and is found in 5 times the concentration in fatty tissue as in the body fluids. It assumes importance only under certain conditions in aviation and diving, or working below sea level.

Respiration in Anesthesia

Before concluding the physiology of respiration it seems worthwhile to remind you of some of the factors which may interfere with ventilation in connection with anesthesia and surgery. Stephen et al[15] have summarized these recently. The first is the presence of preoperative disease, such as asthma, tuberculosis or emphysema. These conditions interfere with getting air in and out easily or with exchanges across the alveolar membranes.

Since one of the most common pulmonary disorders encountered in elderly patients is emphysema, a brief review of this condition will be presented. It is characterized by overdistention of the alveoli, with thinning and rupture of the alveolar walls and loss of elastic tissue. There is reduction in ventilation and both the total lung capacity and the residual volume are increased. The distribution of gases within the alveoli is very uneven, and abnormalities of blood gases are common. The work of breathing is greatly increased, and dyspnea, cough and wheezing are common. The changes in the walls

of the small bronchi and bronchioles narrow the airway and increase markedly the resistance to air flow[16]. The pathologic physiology of pulmonary emphysema has been ably reviewed by Guerrant[17].

The other factors which may interfere with ventilation during anesthesia follow. Preoperative medication, consisting of sedatives and narcotics, depresses the sensitivity of the respiratory center. It does not respond to the normal chemical stimulus (carbon dioxide tension of arterial blood) and nerve impulses as readily as before. Anesthetics and analgesic drugs further depress the sensitivity of the respiratory center. The muscle-relaxant drugs interfere with the transmission of nerve impulses across the myoneural junction and thus paralyze the muscles of respiration.

The operative position itself, the use of rests, the obesity of the patient, large and pendulous breasts may interfere with the efficiency of pulmonary ventilation. The partial immobilization of the diaphragm by packs presents an obstacle to ventilation.

The absorption of carbon dioxide may be imperfect, the endotracheal tube may be kinked or partially blocked, or the mechanical dead space may be unduly large, and all of these factors can impede ventilation. The anesthetist may be inadequate in the assistance of, or control of, pulmonary ventilation.

There is no such thing as "routine" anesthesia or "routine" control of respiratory exchanges. Each patient presents a challenge to the anesthetist.

Postoperative Ventilation

Although postoperative ventilation is not strictly the concern of the anesthetist, there are some hospitals in which the anesthetist is called to consult with those in charge if any difficulty arises. The most common causes of impaired ventilation in the postoperative period are: partial obstruction, depression of respiration due to various drugs, prolonged effects of muscle relaxants, habit apnea, atelectasis, pneumothorax, tight dressings and painful respiration[18, 19]. Partial obstruction may be due to laryngeal edema, tracheal compression or the falling back of the tongue. The depression of respiration may be long-lasting because drugs and anesthetics may be eliminated slowly. Habit apnea may occur after long periods of controlled respiration, and it sometimes lasts as long as 15 minutes. It is not enough to bring a patient safely through the period of anesthesia; the anesthetist should do all in her power to make the patient able to ventilate efficiently by spontaneous respiration as soon as the operation is over.

Implications of Respiratory Physiology in Relation to Air Travel

I have been asked to include "some discussion of the implications of respiratory physiology in relation to air travel." In the first place, flying involves living for a time at a higher altitude, where the oxygen tension is lower than at sea level. Even if one flies in pressurized cabins, the oxygen tension is lower than at sea level. A Douglass DC 6 which cruises at an altitude of 20,000 feet maintains a cabin altitude of about 8500 feet, and a Boeing Stratocruiser at a cruising altitude of 25,000 feet maintains a cabin altitude of 5000 feet[20]. A normal individual notes no symptoms of low oxygen tension below 10,000 feet, but a particularly susceptible

individual, whether pilot or passenger, can develop symptoms of hypoxia at lower altitudes. We shall see later what happens to an air traveler when subjected to hypoxia, keeping in mind that many of the same things may happen to a patient in the operating room when hypoxia supervenes.

In the second place, the air passenger may be frightened, especially if it is the first flight. But even seasoned travelers and pilots, as well, may become frightened when something seems to be going wrong or when a severe electrical storm occurs during flight. It is remarkable how quickly a strong emotion such as fear can affect respiration. One of the first responses to emotional stress is hyperventilation[21]. Hyperventilation produces many changes in the body, which are to be discussed later. The effects of hyperventilation are important not only in aviation, but also in the operating room whenever you hyperventilate a patient whose respiration you are assisting or controlling.

In the third place, there are occasions when sudden ascents are necessary, and there may not be time to pressurize the cabin. Accidental sudden decompression sometimes occurs. In such circumstances, dysbarism (aeroembolism) may occur, but this is far less common than hypoxia and hyperventilation. The characteristics of this condition will be discussed later.

Before discussing hypoxia, hyperventilation and dysbarism, we shall consider briefly "who may fly", from the respiratory standpoint. Should anyone who is permitted to travel at all be allowed to fly?

So far as asthmatic patients are concerned, there is no definite answer at present[22]. Air travel is probably possible in pressurized aircraft, if no more than minimal asthma is present. The crucial altitude seems to be 8000-10,000 feet.

Many patients with severe pulmonary disease have been safely transported by air[23, 24]. However, in severe emphysema there may be rupture of blebs, with subsequent pneumothorax[25].

It is probable that patients with tuberculosis should not fly above 10,000 feet as shown by the subsequent long range course of the disease[26]. Active cases and those with pneumothorax should not fly[20].

Patients with cancer of the lung, pulmonary abscess, and bronchiectasis may fly if oxygen is available and if they are not dyspneic on slight exertion. Pneumonia patients, if comfortable at rest, may fly, if oxygen is available. Patients with foreign bodies in the lung may fly if oxygen is available, but they should be deplaned as soon as possible if pulmonary edema occurs[20].

Many patients with poliomyelitis have been transported safely by air, even in respirators, which, of course require very special care[27].

Physicians now hesitate to permit anyone to fly who has wired jaws because of the possibility of vomiting and aspiration[28].

Individual physicians have to decide whether or not patients with pulmonary disorders may fly. When there is a question, the medical directors of the various airlines may be consulted.

We shall now return to a consideration of the characteristics of hypoxia, hyperventilation and dysbarism.

A. Hypoxia. Hypoxia is characterized by low oxygen tension in the alveoli and/or in the arterial blood.

There are several types of hypoxia, which have been given names descriptive of the causes. The most common is the anoxic type, and it is most often due to ascent to high altitudes without inhalation of oxygen. The composition of air is the same as at sea level, but atmospheric pressure is lower. At 18,000 feet, the oxygen tension is about one-half that at sea level. At 37,000 feet an individual is hypoxic, even if he is breathing 100% oxygen; at this altitude oxygen must be administered under pressure to prevent hypoxia.

poisons such as sodium cyanide. The enzyme systems of the cells are destroyed, and although oxygen is available, the cells are quite unable to make use of it. The various types of hypoxia are illustrated in Figure 8. The end result in all types of hypoxia is the same: the cells have not enough oxygen to utilize the foodstuffs to supply energy for the vital functions.

Various stages of anoxic hypoxia have been described[29]. The first is called the indifferent stage or mild hypoxia. The only effect noted is the impairment of dark adaptation, which

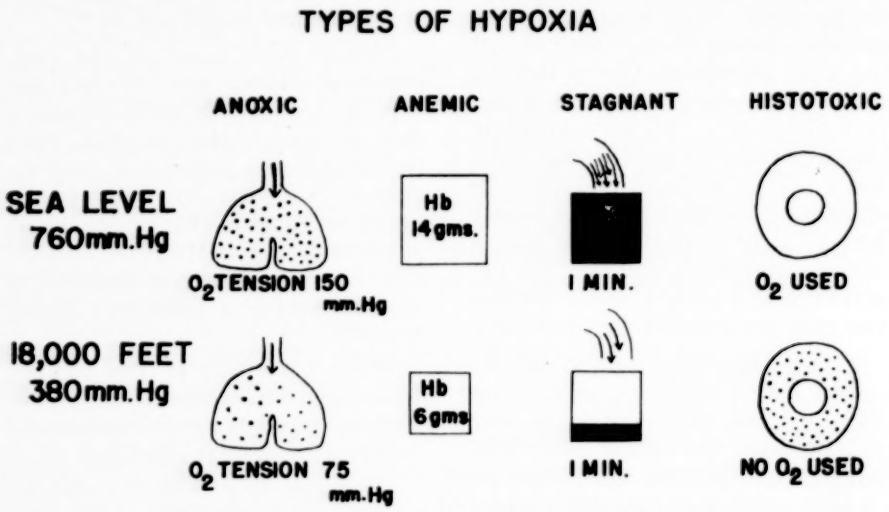


Figure 8. A diagram illustrating the various types of hypoxia.

The second type of hypoxia is anemic hypoxia; in this condition there is too little hemoglobin to transport the required amount of oxygen.

The third type is stagnant hypoxia. This is due to impaired circulation; it is common in heart disease. The circulation is so slow that oxygen is not brought rapidly enough to supply the demands of the tissues.

The fourth type is histotoxic hypoxia; this occurs after the intake of

interferes with night vision and may cause difficulty with pilot efficiency.

The second stage has been called the compensatory stage. In this, respiration is increased in both frequency and depth. The pulse rate, systolic blood pressure, rate of circulation and cardiac output are all increased.

The third stage is called the disturbance stage. The subjective symptoms are fatigue, lassitude, somnolence, vertigo, headache, breathless-

ness and euphoria. The objective signs are impairment of vision, touch, pain and hearing. There is also intellectual impairment: the thinking is slow, calculations unreliable, memory faulty and judgment poor. The reaction time is delayed. There may be a release of undesirable basic personality traits and emotions. Muscular coordination is poor. Both hyperventilation and cyanosis may be present.

The fourth stage is the critical stage. In this, consciousness is lost. This may be the result of the circulatory failure or damage to the central nervous system. There may be convulsions.

With hypoxia there may be increased permeability of the capillary membranes, with edema of various tissues. Edema of the brain leads to headache, nausea and vomiting.

It is evident that hypoxia affects many of the reactions of the body. Probably the most serious consequence of hypoxia in aviation, as well as in anesthesia, is permanent damage to vital organs, such as the brain, heart and kidneys.

B. Hyperventilation. Any individual can bring on the symptoms of hyperventilation by voluntary forced breathing. If he forces inspiration and expiration at an approximately nor-

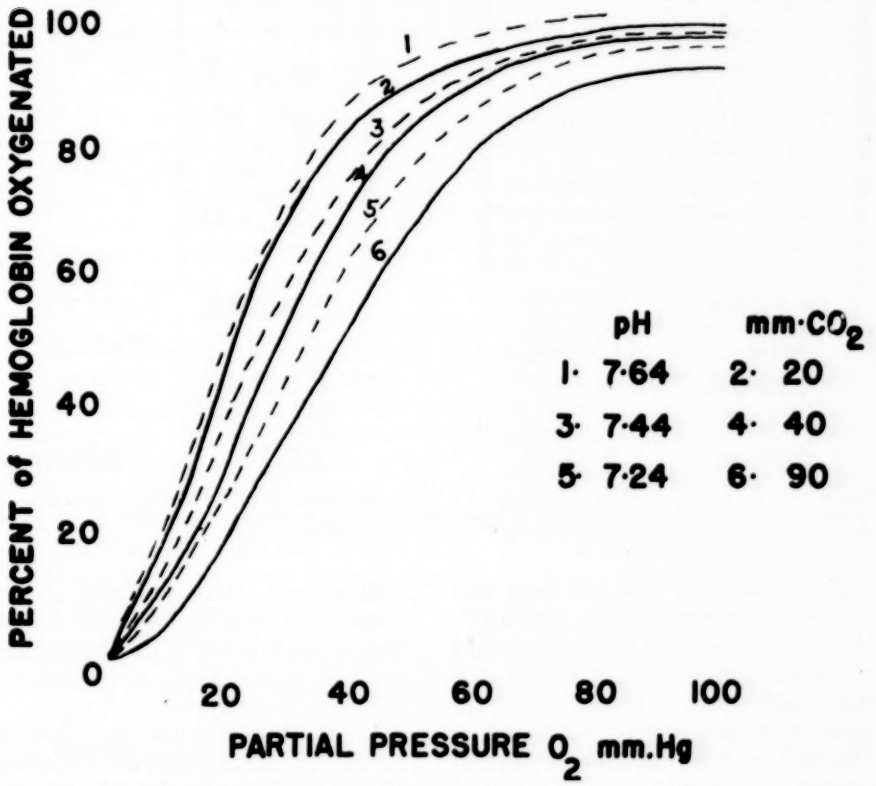


Figure 9. A diagram to show the effects of hyperventilation (respiratory alkalosis) on the oxygen dissociation curve. With the blowing off of carbon dioxide (lowering its tension to 20 mm. Hg) and the rising alkalinity (pH 7.64), the hemoglobin becomes reluctant to part with its oxygen and hypoxia occurs, despite hyperventilation.

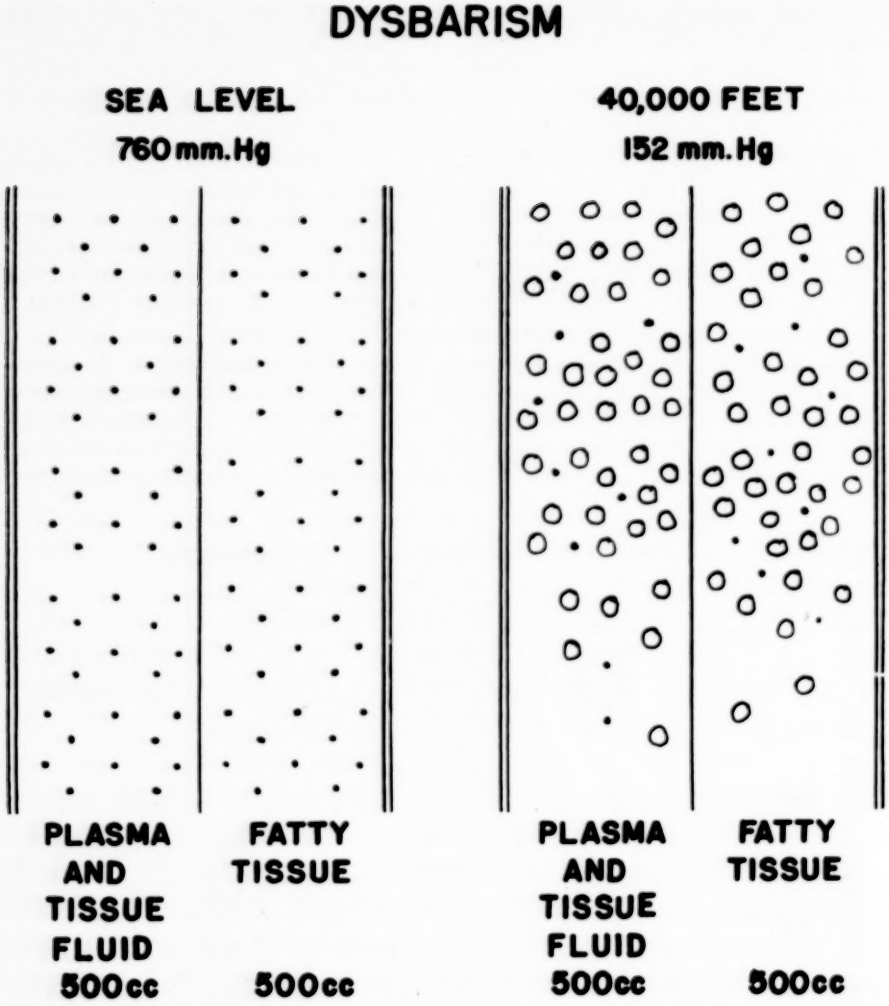


Figure 10. A diagram to show the effects of a very rapid ascent to 40,000 feet in a non-pressurized cabin. Four-fifths of the nitrogen tends to come out of solution in the form of bubbles of gas, due to the suddenness of the change from sea level to 1/5 of an atmosphere.

mal frequency, he will feel faint and dizzy within 30 to 60 seconds[30]. Within a minute or two he will feel numbness and tingling in his extremities, and he may have a sense of panic and palpitation. His mind is clouded, and he borders on unconsciousness. If he continues forced breathing for 5 minutes or more, the muscles become tense and tetanic contractions (tetany) may occur. There may be a feeling of air hunger and of constriction in the thorax. The pulse is weak, rapid and irregular. The skin is pale, cold and moist.

During hyperventilation, when the carbon dioxide is blown off, the hemoglobin holds on to the oxygen with more tenacity and the individual may really suffer from hypoxia, due to the

effects of low carbon dioxide tension, despite good oxygen saturation[31]. The dissociation curve of oxyhemoglobin is displaced to the left, as shown in Figure 9. Both hypocapnia and hypoxia are hazardous conditions, and when they exist simultaneously their effects are additive.

It is sometimes difficult to differentiate between hypoxia and hyperventilation clinically. Many symptoms of the two are alike[32] and one may lead to the other condition. The EEG patterns are similar in the two, which means that they exert similar effects on the electrical activity of the cerebral cortex.

So far as blood chemistry is concerned, there is alkalosis, despite high blood levels of lactic acid. Even the brain has a high content of lactic acid. This may be due to the decreased cerebral blood flow due to vasoconstriction which accompanies hypocapnia. The respiratory alkalosis of hyperventilation leads eventually to metabolic acidosis. The pathway of transformation from one to the other is still a moot point[33].

C. Dysbarism (aeroembolism). At sea level the partial pressure of nitrogen is about 550 mm. Hg. in alveolar air. This is in equilibrium with the blood. The solubility of nitrogen is such that about 1.4 volumes % are dissolved in the plasma. At sea level there is about 1 liter of nitrogen dissolved in the body fluids and in the fatty tissues of the body. Since the solubility is greater in fatty tissue than in water, about 500 cc. are held in fluids and the same amount in fat, which composes less of the body weight than fluid.

If one ascends rapidly from sea level to very high altitudes in a nonpressurized cabin, the nitrogen tends to be released from solution as bubbles of gas. This is illustrated in Figure 10.

One of the common effects of the release of nitrogen bubbles is "bends". In this condition there are bubbles of nitrogen in the connective tissue around bones, joints and muscles. There is deep pain in the extremities.

A second effect of nitrogen bubbles is "chokes". In this condition there are intravascular bubbles in the pulmonary circulation. There is a burning substernal sensation, and there may be cough and a sense of suffocation and apprehension[29].

The treatment of dysbarism is prophylactic; denitrogenation should be carried out by inhalation of pure oxygen at ground level for 15 minutes before flight. The individual susceptibility to dysbarism varies widely.

It is time to close; it would require many hours to treat all phases of respiratory physiology, consequently only these parts which are engaging the attention of clinical physiologists at the present time have been presented in some detail. So far as air travel is concerned, those topics which bear directly on the physiology of respiration have been presented.

The author wishes to express her deep and sincere appreciation to Miss Dorothy W. Ellis who willingly and efficiently drew the illustrations.

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The Circulation in Relation to Newer Technics in Anesthesia

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The Physiology of Circulation

The circulation of the blood has as one of its prime functions that of internal respiration. This is accomplished by the transport of oxygen from the lungs to all the living cells of the body, and carrying carbon dioxide from these cells back to the lungs. Among the many other functions of the circulation are the transport of dextrose, protein and fat from the intestinal tract to their sites of chemical change into other products, or storage, or metabolism; the transport of old red cells to the spleen, lymph nodes, bone marrow, and liver where they are destroyed and transformed into bile pigments, the transport of bile pigments to the liver; the transport of the constituents of hormones to the glands of internal secretion and from these glands the hormones to the sites of their actions; urea from the liver and reticuloendothelial system to the kidneys for elimination. The important functions of circulation to the anesthetist are those concerned with the transport of oxygen, carbon dioxide, dextrose and certain of the hormones.

External respiration has profound effects upon circulation. The most important of these effects are concerned with carbon dioxide, oxygen and the thoracic and abdominal pressures.

The evidences of adequate circulation are not completely satisfactory. Among the factors which help us decide whether circulation is adequate are blood pressure, pulse, heart sounds, consciousness, the electroencephalogram and the electrocardiogram. All of these give us only indirect information as to tissue perfusion.

Circulation

Trying to determine whether circulation is adequate or not is as difficult as assessing the depth of anesthesia with the use of the newer agents.

The blood pressure affords more information about the circulation than any other single observation. The simplest apparatus, which consists of a blood pressure cuff and a stethoscope, is the easiest one to use and is satisfactory under most circumstances. Many other devices have been developed to give more accurate readings of blood pressure. The most important of these is the Statham gage which, by means of a catheter placed in an artery, gives direct readings of blood pressure which can be

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either visualized on an oscilloscope or preserved as a permanent record by a penwriter. A simple device which affords continuous observation of intra-arterial blood pressure consists of an aneroid manometer connected to the intra-arterial catheter.

It is difficult to estimate a safe blood pressure for an individual patient. A systolic blood pressure of 80 mm. of mercury may be adequate for a usually normotensive individual for limited periods during anesthesia. Hypertensive patients may safely tolerate a fall of 30 to 40 per cent from their usual pressure.

The pulse alone gives accurate evidence of the heart rate except in auricular fibrillation, and much information on the force of the heart beat. The quality of the heart sounds is a valuable indication of the action of the heart, especially in children. The consciousness of the patient under spinal anesthesia is a satisfactory indication of adequate cerebral blood flow and perfusion of the brain. The electroencephalogram in the unconscious patient is almost as satisfactory. The electrocardiogram, however, should not be depended on as an evidence of satisfactory cardiac function as strikingly good tracings may be seen in patients with almost no blood pressure or who are almost at the point of death. It does, however, provide valuable knowledge on the integrity of the conduction mechanisms and on the irritability of the myocardium of the ventricle.

Homeostatic Mechanisms

The blood pressure, the cardiac output, and the degree of peripheral vaso-constriction, which are the essential elements in adequate circulation, are normally controlled within narrow limits by the proprioceptive automatic homeostatic reciprocal servo controls. The baro- and chemoreceptors, and the vasomotor, cardiac, and respiratory centers located in the medulla oblongata in the floor of the fourth ventricle are of most importance in circulatory homeostasis. In addition, certain substances act directly on the peripheral blood vessels to control their caliber and thus influence peripheral vascular resistance.

Blood pressure depends upon the cardiac output, the circulating blood volume, the viscosity of the blood, the elasticity of the blood vessels, and the peripheral resistance. The cardiac output depends upon the venous return, the strength of the contraction of the heart, rate, and the arterial pressure.

The elasticity of the arteries is a fixed factor and is not susceptible to change by the surgeon or the anesthetist. The viscosity of the blood may be increased during operation through blood concentration following the loss of large quantities of plasma which may occur most typically in burns. The blood volume in most operations does not change enough to be a significant factor in blood pressure. However, it is of great importance in patients who have lost large quantities of blood through accidental trauma or in operations in which considerable blood loss is unavoidable.

The peripheral resistance of the vascular tree may change greatly during operation and anesthesia. In light anesthesia the homeostatic mechanisms are still active and may effect rapid changes in the capacity of the vascular tree by causing dilatation or constriction of the arterioles. During anesthesia in which the

homeostatic mechanisms are depressed or lost the anesthetist has at hand many agencies and drugs for controlling with some degree of accuracy the peripheral resistance.

The cardiac output during anesthesia may be influenced by favoring venous return to the heart, stimulating the heart muscle to greater activity by drugs such as epinephrine and digitalis, by increasing the heart rate (atropine) and by the artificial control of peripheral resistance.

The Vasomotor Center

The vasomotor center controls the caliber of the smaller blood vessels in three ways. First, it sends a continuous stream of impulses through the sympathetic nerves to the smooth muscle of the blood vessel, causing constriction. A normal flow of these impulses keeps the blood vessels in a state midway between constriction and relaxation. The decrease in the flow of these impulses results in vasodilatation, and an increase, in vasoconstruction. Secondly, the vasomotor center sends impulses through the sympathetic nervous system to the adrenal gland which stimulates the secretion of adrenalin which in turn causes a constriction of the blood vessels. Thirdly, impulses from the vasomotor center travelling by way of the para-sympathetic nervous system over the craniosacral nerves causes vaso-dilatation in restricted areas of the body.

The vasomotor center receives stimuli from many sources. The higher cerebral centers when disturbed by emotion or pain send impulses which pass to the vasomotor center which then may produce either a rise or a fall in blood pressure. It is well known that strong emotion may cause a fall in blood pressure which results in fainting and, on the other hand, painful stimuli result in a rise in blood pressure. This latter is one of the common causes of a rise in blood pressure during surgery under light anesthesia. Carbon dioxide excess and anoxia act on the carotid and aortic bodies and on the vasomotor center to increase peripheral resistance. Carbon dioxide acts chiefly on the vasomotor center and anoxia on the chemoreceptors (the carotid and aortic bodies). Changes in blood pressure are sensed by baroreceptors in the carotid sinus and the aortic arch and pass to the vasomotor center by way of the sinus and aortic nerves via the glossopharyngeal nerve and the vagus nerve respectively. Impulses from the carotid sinus and aortic arch use the same pathways. This is probably the most important mechanism in the homeostatic control of blood pressure. Impulses from these baroreceptors also pass to the cardiac center and thus have an influence in controlling the cardiac output

The Cardiac Center

The cardiac center is similar to the vasomotor center in that it receives impulses from several sources and dispatches stimuli of two kinds to the heart by means of two pathways. Impulses from the higher centers may cause wide variations in the heart rate. Impulses from the respiratory center to the heart center produce sinus arrhythmia. Messages from the carotid sinus, the right auricle, and the arch of the aorta reach the cardiac center by way of the sinus and aortic nerves. These buffer nerves, as they have been called, are of the greatest significance in maintaining arterial blood pressure within normal limits throughout the body and particularly in the brain and the coro-

nary arteries. The stimuli from the cardiac center which either slow or speed the heart travel by the sympathetic and the vagus nerves respectively.

The heart rate is also influenced by agents which act upon it directly. A lack of oxygen of moderate degree and a mild excess of carbon dioxide cause an acceleration of the heart rate. Anoxia and hypercarbia of greater degree will ultimately cause slowing and cessation of the heart beat (from heart block) by causing a depression of the sino-auricular node and the conducting mechanism of the myocardium.

Carbon dioxide when elevated in the venous blood increases extensibility of the myocardium in diastole. The junctional tissues of the heart are depressed by elevated carbon dioxide, AV conduction is depressed and complete heart block results at a pH of 7.0. A rise in the temperature of the blood increases the pulse by stimulating the sino-auricular node.

Humoral Control of Peripheral Resistance

The size of the peripheral blood vessels is controlled by local mechanisms as well as by the nervous impulses from the vasomotor center. Adrenaline, noradrenalin and pituitrin cause vasoconstriction while histamine, metabolites, carbon dioxide excess, oxygen lack and elevated temperature cause vasodilatation.

Effect on Circulation of Newer Anesthesia Techniques

Light Anesthesia

The widespread trend toward lighter anesthesia for surgery of all kinds may be exemplified by three specific technics, namely, ether analgesia, nitrous oxide and hyperventilation, and a combination of general anesthesia and a muscle relaxant.

Ether analgesia as used and described by Artusio[1] has been successful in such operations as open heart surgery. The patient is carried to surgical anesthesia by the use of ether anesthesia, after which the depth is lightened until, during the surgery, including operation upon the heart itself, the patient is able to communicate with the surgeon by opening and closing the eyes and nodding and shaking the head. There is, however, complete amnesia covering the period of surgery. Nitrous oxide and hyperventilation (the dangers of which will be mentioned later) may be used to provide a similar analgesia. In both of these technics as used for cardiac surgery muscular relaxation is not of as great importance as it is in operations upon the biliary tract for example, since the surgical field is well exposed by means of the self retaining rib retractor. General anesthesia by any of the commonly used agents combined with a muscle relaxant and artificial respiration provides nearly ideal conditions for many types of surgery, including those which demand a high degree of muscular relaxation. These three technics have in common the possibility of light anesthesia. Wide experience and careful attention to the patient help to lessen complications. The disadvantages of light anesthesia are three: the patient may move during the operation thus annoying the surgeon, he may suffer the pain of the surgery and remember it afterwards, and he may exhibit changes in blood pressure either a rise or a fall. It is often difficult to assess the degree of amnesia of a patient who, when lightly anesthetized, is still able to respond

to questions and commands. When he cannot respond because of paralysis by one of the muscle relaxants it is nearly impossible to be certain that he is not feeling pain. These complications are not a serious threat to the patient's life but might be the basis for a lawsuit.

The changes in blood pressure caused by pain in a lightly anesthetized patient may be annoying to the anesthetist and dangerous to the patient. Sharp pain of moderate duration raises blood pressure through the spinothalmic and cerebro-cortical connections. Prolonged severe pain may cause a serious fall in blood pressure as a result of strong stimuli from the higher centers. Treatment of these aberrations in blood pressure consists in deepening the anesthesia and the use of an analgesic agent such as Demerol®.

HYPO- AND HYPERVENTILATION

It is customary and necessary to provide assisted or controlled respiration in many of the anesthesia technics in use today. It is as difficult to determine whether a patient is adequately ventilated, without either hypoventilation or hyperventilation, as it is to evaluate an adequate circulation or the depth of anesthesia. Moderate hyperventilation is preferable to hypoventilation and the manner in which it is used in most operations is apparently without danger. Severe hyperventilation, however, results in electrolyte imbalance, particularly a fall in plasma potassium which may result in a serious impairment of cardiac action[2].

DRUGS

The drugs to be considered here include those used for anesthesia and those used for other purposes preceding or during anesthesia and surgery. Among the anesthetic drugs to be discussed are the muscle relaxants, Fluothane® and lignocaine. The muscle relaxants most commonly used today are d-tubocurarine chloride and succinylcholine chloride. The curare preparation causes a fall in blood pressure as the result of histamine release and ganglion blockade which is usually not serious. Although curare causes partial paralysis of the sympathetic ganglia, the blood pressure fall accompanying its use is probably due also to relaxation of the skeletal muscles which ordinarily support the veins, and even more to the release of histamine[3].

An experiment on dogs showed that the splanchnic ganglia were only one-fifth as sensitive to the action of curare as were the skeletal nerve endings of the gastrocnemius muscle[4]. Occasionally, and especially in ill patients, a fall of as much as 30 mm. of mercury in systolic pressure may result from the use of ordinary doses of curare.

Succinylcholine chloride occasionally causes a moderate rise in blood pressure, as does also Flaxedil®. The greatest danger in the use of the muscle relaxants is that of hypoventilation and ensuing anoxia and hypercarbia (asphyxia). This may be a threat to the life of the patient not only during surgery but also and perhaps more particularly in the postoperative period. It is not sufficient to supply enough oxygen to the patient; it is also important to remove carbon dioxide. The patient should be ventilated with approximately eight liters per minute. Air is as satisfactory as oxygen and may be administered by gas machine with pressure on the bag, or by a respirator. The dangers of asphyxia include per-

manent damage to the brain, cardiac arrest, metabolic acidosis, and injury of the kidneys and liver.

One of the greatest dangers in anesthesia today is unrecognized respiratory acidosis from CO_2 retention which is very difficult to recognize by any clinical means[5].

It is generally conceded that a patient who is respirated with an atmosphere containing twenty per cent oxygen with a tidal exchange of 500 cc. 16 times a minute is adequately ventilated. Observation of the bag on the gas machine cannot be expected to give accurate information as to the tidal exchange. The bag ordinarily used is one of 5 liters capacity and observation alone does not afford sufficiently accurate information as to whether each breath is 500 cc. The difference between a breath of 300 cc., which may be inadequate, and 500 cc. which may be adequate, is 200 cc., or forty per cent. For this reason, it is customary to be sure the patient breathes enough by using moderate hyperventilation. The sudden correction of hypercarbia which results from hypoventilation may result in so-called cyclopropane shock or in ventricular fibrillation.

Fluothane anesthesia is one of the newer anesthetic technics which may produce profound hypotension and bradycardia by its direct action on the myocardium. In addition, it sensitizes the myocardium to the action of certain of the pressor amines, the combination of which may result in ventricular fibrillation.

Lignocaine (Xylocaine®) has been used in a recent study as a supplement to general anesthesia. It had been noted earlier that regional anesthesia carried out by the use of lignocaine frequently resulted in central depression of the patient which resulted in sleep. This drug has been used in doses of 250 mg. with half the normal preoperative medication in order to reduce the quantity of general anesthetic agent required. The lignocaine was repeated during the procedure in the same dosage at intervals of one hour. The effects of lignocaine on the circulation are seen with the use of large doses or in unusually sensitive patients, and are evidenced by fall in blood pressure and occasionally cardiac arrest. The effect of the drug is on the conduction mechanism of the heart and the myocardium[6].

Drugs other than anesthetic agents used before or during surgery and anesthesia include hypotensive agents, Regitine®, tranquilizers, steroids, antibiotics, and potassium citrate. All affect the circulation to some degree.

Arfonad®, hexamethonium, and nitroprusside have been used to reduce the blood pressure during surgery and anesthesia. The chief action of hexamethonium is upon the sympathetic ganglia, Arfonad® acts upon these ganglia and the blood vessel itself, and the action of nitroprusside is confined to the blood vessel. All the dangers of hypotension, hypoxia and hypercarbia accompany the use of these agents and they are recommended, if necessary, for short periods only, during which unusual attention must be devoted to the ventilation of the patient. The effects on circulation may be profound as they may reduce blood pressure below the limits at which it can be read.

Regitine® is a potent antiadrenergic agent which blocks the peripheral effects of sympathetic nerves. It is used in surgery of pheochromocytoma to prevent and treat the severe hy-

potension which may result during manipulation of the tumor which results in the release of large amounts of adrenalin and noradrenalin. The patient's blood pressure must be constantly observed, and must be adjusted to within normal limits by injections of Regitine® during surgery and of noradrenalin if necessary after removal of the tumor.

Tranquilizing agents. Agents such as chlorpromazine, Phenergan® and Reserpine®, when used in the days or weeks preceding operation, may be the cause of a highly refractory hypotension during anesthesia. Chlorpromazine, in doses of 25 mg. given to ten male patients scheduled for operation, was found to reduce the effective circulating blood volume in seven[7]. Reserpine has been found to cause such a marked interference with the action of noradrenalin that the patients who received it seemed to act as if they had been sympathectomized[8, 9].

Care must be taken to elicit if possible the history of the use of these drugs before anesthesia is started.

Steroids. The use of steroids such as cortisone and hydrocortisone or ACTH before surgery may result in evidences of adrenal failure with alarming and refractory hypotension during surgery and anesthesia, if these drugs have been discontinued shortly beforehand. An intravenous preparation of hydrocortisone should be available for correcting this deficiency, if necessary.

Antibiotics. Some of the mycins, notably neomycin, have been found to cause a profound muscular relaxation with apnea following anesthesia. One case is reported in which neomycin was introduced into the peritoneal cavity before closure following resection of the ileum in a five day old child. Spontaneous breathing did not reappear and artificial respiration was carried out for a period of two hours. At this time, calcium was given intravenously, whereupon the spontaneous respirations reappeared. A few days later, another laparotomy was necessary on account of intestinal obstruction and the same anesthesia was carried out with the exception of the neomycin, and no respiratory difficulty followed.

Potassium citrate. In the course of operations upon congenital defects of the heart, the contractions of the myocardium are arrested by the perfusion of the coronary arteries with a solution of potassium citrate. The potassium ion prevents muscular contraction and the citrate ion, by combining with the ionized calcium, produces the same effect. A completely flaccid heart with no sign of activity on the electrocardiogram results. At the end of the procedure, which lasts up to an hour in some instances, the heart beat is restored by allowing blood from the pump oxygenator to flush out the potassium citrate from the coronary arteries and the myocardial cells and to restore normal quantities of calcium.

The use of induced hypothermia, the artificial kidney, and mechanical respirators may have marked effects upon the circulation.

INDUCED HYPOTHERMIA

The refrigeration of a patient results in slowing of the heart rate and fall in blood pressure as a result of the depressing effect of cold upon the vital centers and the myocardium.

THE ARTIFICIAL KIDNEY

The necessity of removing blood from a patient's artery, passing it through an artificial kidney and re-

turning it to a vein for the purpose of removing nitrogenous products, or such drugs as certain of the barbiturates and aspirin, may interfere with the normal circulation of the patient. The method of local heparinization of the blood in which a slow drip of a heparin solution is mixed with the blood as it leaves the patient and the neutralization of the heparin as the blood returns to the patient, results in some cases in a disturbance of the patient's clotting mechanism so that post-procedure hemorrhage may follow. Furthermore protamine itself may cause a serious fall in blood pressure if administered too rapidly.

RESPIRATORS

Respirators as used in the operating room and in the immediate postoperative period, although they may be the cause of a moderate hyperventilation and hypocarbia, do not ordinarily lead to serious results. Intermittent positive pressure alone may decrease cardiac output by reducing venous return to the heart. Furthermore, in patients such as the victims of poliomyelitis whose respiration has to be sustained by artificial means for many weeks or months, the pH and carbon dioxide content of the blood must be followed with great care and preserved within normal limits to avoid electrolyte imbalance from overventilation.

SURGICAL PROCEDURES

1. Massive blood loss and transfusion. Patients who lose large quantities of blood through accident, or during surgery accompanied by uncontrolled hemorrhage, may suffer disturbances in circulation through many of the mechanisms mentioned above. Blood replacement should be carried out rapidly and should keep pace with blood loss when circumstances permit. Massive transfusion sometimes leads to a serious defect in the clotting of the blood which is, as yet, incompletely understood. Rapid transfusion may be followed by myocardial failure secondary to the fixing of ionizable blood calcium by the citrate in bank blood. Calcium gluconate should be administered to patients who receive transfusions of more than 3 units of blood in a short period of time.

2. Position. The prone position as used for laminectomy and the Kraske or jackknife position, used in perineal operations or for the posterior resection of the rectum, may cause several disturbances in circulation. Pressure on the abdominal viscera causes an elevation of the diaphragm which is followed by hypoventilation, anoxia and hypercarbia. Pressure on the inferior vena cava, which is much more readily collapsed than the aorta, may result in the trapping of a considerable quantity of blood in the lower extremities which, for the time being, acts as a temporary hemorrhage of this amount. These possibilities must be borne in mind and especially in poor risk patients another position should be chosen or the position should be changed even during surgery if serious circulatory failure appears.

CONCLUSION

In anesthesia today it is difficult to assess circulation, ventilation and consciousness. Experience and close attention to details during anesthesia are in some measure helpful.

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Anesthesia in Relation to Cardiopulmonary By-pass with Description of a Basic Technique

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Interruption of vital circulatory functions for unusual periods of time is the unique problem of cardiovascular surgery, the urgency of which diminishes as one moves peripherally on the circulatory tree. Thus, the disease focus is the most important single factor in determining the surgical attack and, therefore the anesthetic approach. (Fig. I)

Basically three solutions of the problem are available: shunting, hypothermia, and mechanical cardiopulmonary by-pass. A valuable auxiliary measure is hypotension—produced by an agent such as Arfonad.® (Arfonad® may also eliminate certain undesirable vasospastic reflexes, preventing, in many cases, postoperative anuria.[5])

Shunting is a technical problem of surgery and is not germaine to these notes. Hypothermia has a wide field of usefulness both alone and as an adjunct to other measures; however, it has two serious drawbacks: circulatory arrest is subject to critical time

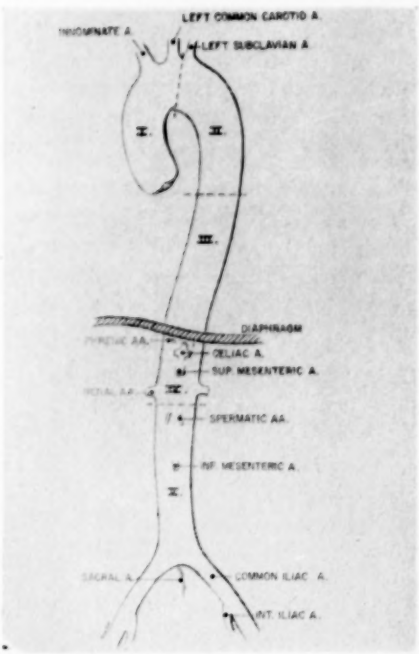

FIGURE I: Techniques applied in the numbered segments are as follows:
Area I (includes heart): Pump oxygenator, shunt, hypothermia.
Area II: Hypothermia, shunt, pump with or without oxygenator.
Area III: Shunt, hypothermia, pump with or without oxygenator.
Area IV: Shunt, hypothermia, pump with or without oxygenator.
Area V: No protection required — cross clamp aorta.
It is felt that area I and II will finally be handled with pump oxygenators while shunts will replace hypothermia.

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limits and the myocardium is disposed to fibrillate.

A properly conducted whole body perfusion permits prolonged, controlled interruption of the circulation at any level. Elective cardiac arrest may be used with this technique to provide a still, bloodless intracardiac surgical field.[4] To achieve the desired effect requires the closest teamwork between anesthesiologist and his assistant; the surgeon and his assistants; the pump operator; and each of these components with one another.[2]

The Extracorporeal Circuit: This consists of a pumping mechanism, an oxygenating mechanism and tubing to connect these various components together and to the patient's circulatory tree. (Fig. II.) Myriad devices are used in refining and elaborating this basic system.[3]

The pumping aspect of a "pump-oxygenator" is exceedingly complex if one considers such controversial factors as pulsatile vs. non-pulsatile flow; occlusive vs. non-occlusive pumps; physical properties of connecting tubing relative to resistance to flow; over-all trauma to blood cells by the various components, etc. Suffice it to say that several practically acceptable pumps are commercially available. It is considered that the best of these to date is the "double roller" type pump.[3]

Methods of oxygenation vary widely:[3] macro bubbling, micro bubbling, filming, and dispersion (Waud principle) are the basic techniques. The bubbling techniques bubble the desired gas mixture through the blood

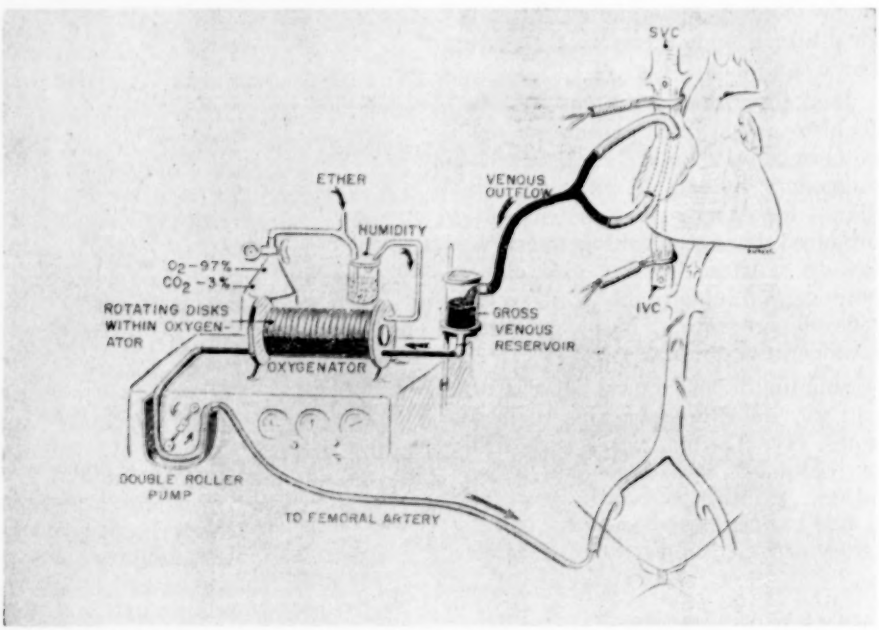

FIGURE II: Basic components of total cardio-pulmonary by-pass. Some essentials are omitted for the sake of simplicity. The major concern here is the tank showing a composite mixture of gases and the humidifying chamber. These will be touched upon subsequently.

in the extracorporeal circuit. While bubbling is traumatic to blood, dispersion is most harmful in that blood is sprayed through an atmosphere of prepared gases. The most reasonable method for oxygenating is based on the filming principle in which a thin film of blood is exposed to a controlled atmosphere. Filming is accomplished in a number of ways: on vertical screens, rotating discs, etc. The most *mechanically* perfect method now available is found in the rotating disc oxygenator. This equipment is, also, reasonably priced.

Anesthetic Aspects: Anesthesia for cardiopulmonary by-pass is little different from any thoracic anesthetic. It is imperative, however, that each detail be carefully executed as the margin for error is practically nonexistent. As a rule a minimum of one anesthesiologist and an assistant is required to properly perform the required functions.

The major considerations are these: the patients are all circulatory cripples and many will require extensive surgical trauma including bilateral thoracotomy.

As a large proportion of these patients will be children, premedication must insure tranquility. The usual Demerol® and atropine are nicely augmented with Phenergan® to achieve this end. Anesthesia may be induced with short-acting barbiturates and muscle relaxants, balanced cyclopropane - ether - curare, etc. The essentials of induction are *rapidity* and *smoothness* to avoid unnecessary neurogenic and metabolic responses. Once induction has been achieved it is essential to maintain the patient at a very light level: more exactly, in a state of analgesia.

The proper anesthetic level is most satisfactorily monitered by the continuous EEG.[7] It may be best to carry these people up to by-pass in stage three, plane one, but this must be lightened prior to by-pass. Control of the patient at this level can be maintained by hyperventilation producing respiratory alkalosis. Hyperventilation is considered essential and has the added advantage in these cases of virtually eliminating the need for muscle relaxants during by-pass proper. Moreover, should anoxia or hypercarbia develop, the relaxants must be given in very large amounts to overcome spasmodic contractions of the diaphragm during by-pass; a highly undesirable situation. Hyperventilation should gain a pH of 7.5 to 7.65 prior to by-pass. This forced respiration requires considerable skill during the closed chest phase as mean endotracheal pressures may be elevated to dangerous levels diminishing cardiac inflow (inadvertent Valsalva maneuver) and, in turn, cardiac output to the point of cardiac standstill.

A physiologic consideration of importance here is the effect of high blood pH and low CO_2 tensions on the dissociation curve of oxyhemoglobin. In other words, a high pH low CO_2 tends to bind oxygen to hemoglobin so that bright red venous blood may be observed even though cellular anoxia and death therefrom may occur (Bohr effect). Also, cerebral vasoconstriction has been postulated to occur under these circumstances. Furthermore, certain cardiac defects prohibit strenuous ventilation by their disastrous response. This is a very technical problem and not well understood at this time. Thus, are recorded the dangers of careless usage of hyperventilation.

Intravenous fluids are undesirable in these patients and are therefore kept minimal. A route of administration for heparin immediately before by-pass and protamine sulfate following by-pass must be maintained. This is done with very little fluid. On the other hand blood loss is measured in detail (by weighing sponges, using graduated suction bottles, etc.) and carefully replaced so that blood volume is correct prior to by-pass.

The anesthetic team will usually have the help of a cardiologist and others during the crucial phases of the procedure.

A Basic Technique: Premedication consists of appropriate amount of Demerol,® atropine and Phenergan.®

Induction is achieved with Surital® and Anectine,® intubation accomplished, and the patient switched to ether (for small children a non-rebreathing technique is preferred; in larger children and adults a semi-closed system is satisfactory). The EEG monitors the desired depth of anesthesia and hyperventilation is begun. As the surgical team approaches by-pass time the pump operator primes and prepares the pump. The anesthesiologist's assistant prepares to vaporize ether into the oxygenator in quantity sufficient to give 3 to 4% concentration of vapor in the oxygenator. This concentration must be determined by prior calibration of the equipment or the use of an apparatus such as the Forreger Copper Kettle. The latter gives a fairly constant amount of ether vapor and charts are available showing the amount of vapor at various flow rates. The pump operator will introduce a gas mixture composed of 97% oxygen and 3% CO_2, well humidified, into the oxygenator at the rate of 6 liters/min.[6] All of these items may traverse a common inlet to the oxygenator. Humidity is important as this lessens the damages to red blood cells which characteristically occurs at a "dry" blood gas interface. Further adjustments are made by the pump operator and by-pass is imminent. The pH is checked, blood volume corrected, and the patient's condition made known to the entire team. If all is well, the anesthesiologist administers heparin intravenously, the patient's heart and blood vessels are cannulated and he is connected for by-pass. At this point the patient is on "partial by-pass". Hyperventilation continues as the extracorporeal circuit is balanced with the patient's circulation.[1] About two minutes of partial by-pass are required following which "total by-pass" is accomplished. The anesthesiologist permits the lungs to deflate approximately 50% and inflates them intermittently (every 90-120 secs.). This inflation must take into account surgical maneuvers as it can flood the surgeon's field with blood.

At the conclusion of intracardiac surgery, partial by-pass is attained and hyperventilation is begun. As the patient is removed from by-pass the pump operator balances the blood volume to make up deficits, decannulation and hemostasis are effected. The anesthesiologist now administers the prescribed antiheparin agent.

From this point practically no anesthetic is given while hyperventilation persists. When the patient begins to move, a mixture of 50% nitrous oxide and 50% oxygen is usually sufficient for the completion of surgery. Every effort is made to have the patient awake by skin closure.

Following by-pass the patient is unusually sensitive to sedatives and narcotics and a fraction of the amount of Demerol® given preoperatively may suffice for 6 to 8 hours.

The patient now requires constant attention for at least 36 hours. Competent surgical and anesthetic teams must be available during this period. Supported respiration may be required up to 24 hours postoperatively. While tracheostomy need not be routine it will frequently be life saving.

Summary

An introduction to some of the anesthetic aspects of cardiopulmonary by-pass is made. A single anesthetic technique is presented.

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A Philosophy of Obstetrical Anesthesia

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The purpose of this paper is to offer a general philosophy of approach to obstetrical anesthesia. A presentation of this sort necessarily reflects personal opinion, and the author realizes that some of the opinions expressed might be subject to dispute. However, it is felt that if the principles herein set forth are rigidly adhered to, anesthesia will be adequate and complications rare.

When this discussion was originally scheduled, the title was listed as *New Trends in Obstetrical Anesthesia*. I asked that it be changed for two reasons. First, I don't believe there are any that are worth discussing at a practical level, so that most of us will for the foreseeable future continue to use methods that are at least 25 years old. Second, I believe that most of the anesthetic tragedies that occur in our delivery rooms happen not because our agents are inadequate, but because they have been improperly used. I am certain that many lives can be saved simply by paying more attention to basic principles which have been well known for many years.

One of the most important facts about obstetrical anesthesia is that it is difficult. The following conditions, common in the delivery room, would be grounds for cancellation of many an elective operation because of anesthetic hazards: (1) a full stomach, (2) upper respiratory infection, (3) obesity, (4) recent major hemorrhage with circulating blood volume severly compromised, and (5) a patient who usually is in pain, and frequently is emotionally tense and physically exhausted. Anesthesia presents no greater challenge than a tired, frightened, fat patient with a nasty cold and a full stomach, poorly premedicated, and suffering the discomforts of the second stage of labor.

In spite of these difficulties, it frequently is the case that the obstetrical department is the orphan child of the hospital — a repository for the patched gloves, the bent Kelly clamps, the needles with the fishhooks on the end, and the anesthesia equipment that is too good to throw away, and too outmoded to use in the operating room. In addition, there are still many hospitals in which no qualified anesthetist is available during all or part of the day. This often results in the nineteeth-century spectacle of an untrained nurse pouring open drop ether, usually with a very poor notion indeed of what she is doing.

The ideal anesthetic to cope with these difficulties has not yet been invented. The perfect agent should have

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the following attributes: (1) 100% safe for the mother, (2) no effect whatsoever upon the infant, (3) easy for the anesthetist to administer and control, (4) pleasant to take, and (5) completely relieve the discomfort of labor and childbirth.

Anyone can look at this list, and pick out serious objections to any of the methods now in use. Each has its own peculiar advantages and shortcomings, and it is my feeling that the best results are obtained by tailoring the anesthetic to fit the particular patient. This implies that on a good obstetrical service, physicians and anesthetists should be prepared to use any of several different methods, and to select the one for the individual case that best answers the situation at hand.

With this as a background, let us then examine individually the more common methods now in use, and try to arrive at a sound basis for deciding which is the best for a particular situation.

Inhalation

General anesthesia, regardless of the particular agent chosen, has certain built-in disadvantages for obstetrics which to me are serious enough to make its use undesirable as a routine procedure. There is the always worrisome question of the full stomach. Many patients will deliver within an hour or two after a large meal. There is no time limit after eating that can be considered safe, for in general when the uterus starts contracting, the stomach stops contracting. A dinner consumed at six o'clock can still be very much in evidence at four a.m. Anyone who has ever had the horrible experience of seeing one good aspiration in the delivery room is not likely ever to forget it.

Aside from the dangers of vomiting, the chief objection to general anesthesia is the danger of fetal depression. Every anesthetic which depends for its action upon reaching a certain level in the mother's blood stream will cross the placental barrier and depress the infant, and the question is only one of degree. This in most instances should not be a major item if the agent is properly administered, and if the over-all interval between induction and delivery is not too great. However, if the limits of prudence are exceeded, or in borderline situations such as prematurity, prolonged labor, cord entanglement, or an infant which is already compromised by excessive premedication, the added CNS depression of a general anesthetic may be the deciding factor between life and death for the infant.

A word should be said about the matter of anesthetizing the patient until the doctor arrives, in order to avoid a precipitate delivery. The practice of calling the physician and anesthetizing the patient when the head begins to distend the perineum cannot be too strongly condemned. Every perineal laceration can be satisfactorily repaired, but the damage to a baby from too much anesthesia can be irreparable. A patient who is ready for delivery should be delivered —by herself or by anyone who is present. Whatever explanations are necessary can be made later.

One other hazard of general anesthesia has been introduced by the recent popularity of trichloroethylene for analgesia during labor. Patients who have been using Trilene® should never be changed over to a closed circuit gas machine, as it is well known that Trilene® becomes very toxic when exposed to soda lime.

Pudendal Block

This is one of the most useful of all anesthetics in the delivery room. It is quick and easy to administer. It is universally applicable, as there are no actual contraindications to its use. It represents the nearest thing to 100% safety in anesthesia. There are no hazards except the one patient in a million who is violently allergic to the particular drug being used, and the occasional inadvertent intravenous injection of the agent. The former is a minimal risk that must be accepted with any drug from aspirin to Xylocaine®; the latter should never happen if the most elementary precautions are observed.

The major disadvantage of pudendal block is that in many instances it may be considered by the patient to be inadequate anesthesia. This is particularly true if premedication has been inadequate, if the patient is unusually excitable, if she is a primipara, if anything more than the easiest outlet forceps is required to effect delivery, or in the 10% or so in whom the block is not completely successful.

This disadvantage of inadequacy can largely be overcome by supplementing with inhalation, and without giving enough gas or ether to compromise the baby or cause the mother to be unable to handle her own vomitus. Especially with nitrous oxide, one can give considerable analgesia and amnesia without loss of consciousness. The combination of pudendal block with 80% nitrous oxide administered only during contractions is one of the safest and most satisfactory methods of obstetrical anesthesia available.

Spinal

Spinal anesthesia is a very useful and very safe method in obstetrics, but one which has often been abused, and the victim of much misunderstanding by physicians, anesthetists, and the laity. I should like to begin by debunking a few popular misconceptions.

Spinal anesthesia properly used: (1) does not cause sudden and unexplained death, (2) does not cause permanent paralysis, (3) does not cause severe and prolonged backaches, and (4) does not cause headaches, of more than a few days duration.

It should be carefully noted that spinal anesthesia does not *prevent* any of these occurrences. Recurrent headaches are a common problem in any post-partum clinic, and are largely due to emotional strain and loss of sleep. The low back pain which is almost universal during the first weeks after delivery is in most instances 50% fatigue and 50% poor posture. It is unfortunate that these common complaints have many times been attributed to a spinal if one was given, and largely ignored if any other anesthetic was used. An extreme example is the following case from my own experience:

Mrs. J. H., aged 18, was first seen in the dependents' clinic of an Air Force hospital. She annuonced that she was two months pregnant. Examination revealed the gestation to be of at least five months duration. When this information was given, she became very upset. It developed that her husband had returned from overseas only two months before, and that someone else had fathered her child in his absence. After a great deal of bitter discussion between them, he agreed not to dissolve the marriage, if she would give up the child for adoption. At the expected time, she had an uneventful delivery under spinal anesthesia. Some twelve hours later, I was called to see the patient because she had complete paralysis and loss of sensation from the waist down.

On questioning it developed that she was unable to bring herself to give up either husband or baby, and was also very poorly prepared emotionally to accept the full responsibilities of either marriage or motherhood. Her paralysis was a means of evoking sufficient sympathy to keep her husband and her baby, and of prevailing upon her mother to join the family group to share the responsibilities of parenthood of which she felt herself to be incapable. Fortunately, I was able to convince her that this was indeed a poor solution to her problem, and she was completely cured in the space of an hour. In many instances, however, with a little less malingering and a little more true hysteria, the problem might have been more difficult. No one would have been able to convince friends and family that the anesthetic was not responsible for her paralysis.

There are of course, as with most things in medicine, exceptions to the blanket statements we have made regarding safety. Most of the exceptions have been due to major and inexcusable errors of technique, which leads us to a list of axioms for spinal anesthesia that should *never* be violated.

1. No ampule of a drug which is intended for intrathecal injection should be sterilized in a liquid bath of any kind. These ampules must be autoclaved.

2. No physician should ever administer a spinal anesthetic who has not been adequately trained in its use. This is an open invitation to disaster.

3. A spinal anesthetic should never be administered unless a trained anesthetist is at the head of the table in constant attendance. Violation of this one rule has been responsible for most of the tragedies that have occurred with spinal anesthesia in obstetrics. I am constantly amazed at the prevalence of the illogical attitude, by people who would never think of leaving a spinal patient unattended in the operating room, that no anesthetist is necessary for the same procedure in the delivery room. If one surveys the proceedings of any maternal mortality committee in the country, the following sequence of events is repeated time and time again:
   a. The anesthetist on call was busy in the operating room.
   b. The patient was ready to deliver.
   c. The attending physician was relatively inexperienced with the method, but a spinal was elected as the best alternative under the circumstances.
   d. After the drug was administered, the circulating nurse was circulating, the scrub nurse and physician were scrubbing, the anesthetist wasn't even on the same floor of the hospital, and no one was watching the patient's blood pressure.
   e. Eventually the discovery is made that there is no blood pressure (by now five minutes have elapsed since the injection) and the patient is not breathing.
   f. This touches off a wild chain of events which includes, in various combinations, venous cut-downs, artificial respiration, intracardiac stimulants, thoracotomy with cardiac massage, traumatic forceps delivery of the infant, and death of mother and baby.

4. One agent or combination of agents should be used by a given individual, and only one. Each drug used for spinal anesthesia has its own peculiarities of behavior, and the physician should become

thoroughly familiar with the vagaries of one drug, and then stick to it.

5. The dose of a given spinal anesthetic for a pregnant woman is exactly half of what would be administered for the same individual not pregnant. Violation of this rule can be fatal.
6. The injection should be made only at the end of a contraction.
7. The blood pressure *must* be checked every minute for the first five minutes, and frequently thereafter. The first five minutes are crucial, and a momentary distraction at this time can have serious consequences. There is no excuse ever for finding a patient with a blood pressure of 0/0, or 40/0. There was a time when that blood pressure was 100/80, and another time when it was 80/60. Correction at that stage would have been a simple matter.
8. A syringe, and vasopressor drug, with which the anesthetist is thoroughly familiar, must be within reach of the anesthetist's chair at all times.
9. An airway, and a machine for giving positive-pressure oxygen, must be immediately available.

I have never seen nor heard of a major mishap with spinal anesthesia when these precautions have been observed. Even the occasional "complete" spinal which goes right up to the ear lobes should not result in any more serious damage than a little wear and tear on the nerves of the anesthetist if the situation is properly handled.

Then if none of the usual objections to spinal anesthesia are valid, what are its disadvantages?

1. As with pudendal, there will be a rare individual with an anaphylatic type of response to the agent being used.
2. There have been a few cases of persistent neurological changes which apparently are bona fide. Most of these complications have been relatively minor and usually transitory. They are quite rare—especially in reported series in which the author has been careful to exclude pre-existing neurologic disease.
3. The problem of headaches is a real one. This almost never lasts more than a few days, and no permanent disability ever results. The complication is strictly in the nuisance category, but it is very annoying to the individual involved, and there is no doubt that it is more common in obstetrical than in surgical patients.
4. In spite of the fact that there *should* be no accidents with the method, it cannot be denied that there is much more room for human error than with pudendal block, for example. So long as people are not infallible, a certain irreducible number of accidents will happen — the same as with blood transfusion, or any other hospital procedure in which a minor violation of routine can have serious consequences.

Caudal

I do not use caudal anesthesia for the simple reason that I do not believe it is as safe as other methods. Even in the best of hands, and with utmost precautions, there have been by now a considerable number of accidental intrathecal injections made by the caudal route. Since the dosage used for caudal is well above the allowable maximum for spinal, the outcome is usually fatal. Unlike spinal anesthesia, these tragedies, while rare,

are inherent in the method, and not necessarily the result of carelessness of technique.

Natural Childbirth

This has been the major fad of the last decade, the subject of innumerable articles in the popular magazines, and the source of a great deal of nonsense in high places. To anyone who has ever dealt with patients it is perfectly apparent that pain—any pain—is exaggerated by fear. This has been a basic tenet of the art of the good physician for centuries. I am heartily in favor of any steps that can be taken—be it prenatal education, fostering a strong bond of understanding between doctor and patient, etc.,—to improve the confidence and tranquillity of the patient. But to say that all obstetrical pain is fear, to make a fetish of using absolutely no medication of any kind, or to suggest that a new race of better adjusted people is being created by the method seems a little beyond the pale of logic. I have yet to be convinced that the recommended series of yogi exercises have any merit other than that of giving the patient something else to think about besides her discomfort.

Aside from this, there is one other objection that seems to me to be potentially serious. There has been an unfortunate tendency for this technique to attract the one personality type who should never attempt it. This is the individual who has a basic feeling of inadequacy to play the role of mother, and who therefore feels a compelling need to do something heroic to prove her capabilities. She is doomed by the nature of her make-up to failure, and is the very patient who should never be allowed to suffer a major defeat in her first experience with parenthood. The women who fit into this pattern are a small minority, but many of them will become major psychiatric casualties of the method unless they are very carefully handled.

Hypnosis

This is destined to be the fad of the next decade, as "natural" childbirth has been of the last. The only objection to hypnosis in obstetrics is that it is not very practical. Approximately 1/3 of the patients are good subjects, and can be prepared with a total of one or two hours of practice during pregnancy. Another 1/3 can be successfully prepared by spending a great deal more time than the ordinary practitioner has to give. The remaining 1/3 either cannot be hypnotized, or the time required in preparation would be so great as to be not worth considering. Accordingly, this would not seem to be a satisfactory answer to the problem of obstetrical anesthesia, at least for a busy practitioner.

Cesarean Section

The special problem of anesthesia for cesarean section deserves some comment. It is my practice to use spinal anesthesia routinely for this procedure, except in cases where there has been major, uncorrected blood loss. In addition to the advantages already enumerated, it allows the surgeon to proceed without violent haste, and therefore to do a more precise and less traumatic type of operation. This is particularly true in the patient who has had previous lower abdominal surgery, wherein adhesions may be a major obstacle. Since local anesthesia is apt to be very satisfactory to everyone but the patient, my preference is for cyclopropane in those cases for which spinal is not suitable.

(Continued on page 60)

Notes and Case Reports

Concurrent Use of Ilopan and Succinylcholine

Ilopan (pantothenyl alcohol, Warren-Teed Products Company) is a principal component of coenzyme A, a factor essential to the formation of acetylcholine, the substance required for normal peristalsis. Pantothenic acid becomes the logical key in cases of postoperative decreased intestinal motility because, very often, the postoperative patient appears to undergo a deficiency of, or increased demand for coenzyme A due to surgical stress and other factors.

Ilopan is indicated for intestinal atony and distention, postoperative retention of flatus and feces, or postoperative delay in resumption of intestinal motility, paralytic ileus and prophylactic use immediately after abdominal surgery to minimize the possibility of paralytic ileus. There are no known contraindications and no reported side effects when used alone to restore normal peristalsis, but neostigmine and other enterokinetic drugs should not be used concurrently with Ilopan because of the danger of overstimulation. If such drugs have been used, Ilopan should not be started sooner than 12 hours after enterokinetic drugs have been discontinued. Narcotic analgesics, antihistamines for nausea and other peristaltic depressants, if employed in larger than ordinary doses, should be followed by an increased frequency of Ilopan dosage for best results. Ilopan is safe and well tolerated when given intramuscularly and it cannot produce more than normal peristalsis, which is a distinct advantage.

For more than a year, Ilopan has been administered almost routinely following abdominal surgery in our hospital, with highly desirable results and no known complications. Recently, the drug was administered to a patient on the operating table who had undergone prolonged surgery and who had received a moderately large dose of succinylcholine which had not, presumably, been completely hydrolyzed. It is certainly not to be expected that this situation will occur frequently. However, since the results were dramatic and unexpected, the following case report has been written in the hope that it will prove interesting as well as beneficial to others who administer anesthesia.

Mrs. X was brought to the operating room for a probable total thyroidectomy. She was 50 years old, weighed 138 pounds, and had never been hospitalized previously. Her thyroid mass was thought to be relatively nontoxic and the B.M.R. reading was interpreted as high normal.

The routine laboratory work was within normal ranges with 13.8 grams of hemoglobin, 41 volumes per cent hematocrit, and a negative urinalysis. Although Mrs. X was markedly apprehensive prior to the day of surgery and stated that she would never leave surgery alive, on the morning of the operation she exhibited no abnormal signs of fear. In the operating suite, just prior to induction, her blood pressure was 140/80, pulse was 88 and respirations were 18.

Mrs. X was inducted using 12 cc. of Sodium Pentothal 2½%, given 30 milligrams of succinylcholine chloride (Quelicin Chloride) intravenously and intubated orally under direct vision. Maintenance was effected by the Shane-Ashman technique, using the flow of oxygen 2000 cc., nitrous oxide 1000 cc., and cyclopropane 400 cc. per minute with Quelicin Chloride 0.1% continuous drip to control the pharyngeal reflex.

Mrs. X exhibited no signs of distress throughout the three hour procedure even though the mass was closely adhered to the trachea and much traction was necessary. Color, temperature, and skin tonus remained good, blood pressure ranged from 120/80 to 132/84, pulse was consistently in the 80's and respirations were controlled at 16 per minute. Total blood loss was estimated at less than 200 cc. and 325 cc. of Quelicin Chloride was necessary to prevent straining on the endotracheal tube.

About 30 minutes prior to the completion of the surgery, Quelicin was drastically reduced and the patient was allowed to resume spontaneous respirations. Recovery from the succinylcholine effect was apparently normal. When the drapes were removed, the patient's stomach appeared grossly distended; a Kaslow tube was inserted immediately and the gastric contents were aspirated. The surgeon ordered 2 cc. of Ilopan intramuscularly "stat" which was given approximately five minutes following the complete cessation of the Quelicin drip. At this time Mrs. X had an active cough reflex; the endotracheal tube had been removed, respirations were 18 per minute with full intercostal movements but diminished excursions, blood pressure was 122/82 and pulse was 82. She had opened her eyes, swallowed several times and there was no indication of any form of shock nor complication at this time.

Fifteen minutes later, Mrs. X exhibited marked respiratory difficulty with tracheal tug, chest retraction, and very obvious inadequate respiratory excursions. Although vital signs were otherwise good and unchanged, the pupils were fixed and widely dilated and no reflex could be elicited by painful stimulus. An immediate laryngoscopy was performed and demonstrated both vocal cords to be active. An oral intubation was performed and the patient was assisted on the anesthetic machine for a total period of one and one-half hours during which time all vital signs were good and unchanged except for respirations. Metrazol 2 cc. was given slowly intravenously to detoxify any exisiting Pentothal with no apparent result or improvement. After one hour, because of the suspected prolonged succinylcholine effect, 500 cc. of fresh whole blood was administered to replenish the plasma cholinesterase and some immediate, though not dramatic, improvement was noted.

After one and one-half hours the patient was extubated and transported to her room, responding to questions and demonstrating temporal, spatial, and personal orientation. A tracheal tug persisted for 6 hours and oxygen was administered nasally for 12 hours, after which time Mrs. X effected a rapid, completely uneventful recovery and was discharged ambulatory on the 7th postoperative day with no apparent lasting ill effects from her ordeal in the operating room.

This case was reviewed thoroughly by both surgeon and anesthetist, separately and together, and no acceptable conclusion could be reached that did not include a consideration of the unaccustomed use of succinylcholine and Ilopan concurrently. The Warren-Teed Products Company was contacted and obligingly supplied all available excerpts and brochures concerning their product, Ilopan. Although the author of this article is fully aware of the negligible value of a single case insofar as accurate conclusion is concerned and the possibility of error (unknown or unrecognized factors are always existent) this article was written for what little value it may possess for your interest and information. All technical information contained in this article was obtained from the Warren-Teed Products

(Continued on page 60)

Insurance

The "Why" of Group Insurance

If you were to go to a store and ask for one can of tomatoes, the price would be more than if you bought two cans of tomatoes. You will note in many stores that merchandise is advertised as "2 for 31¢" or "6 for $1.00". If you were to go to the wholesale store and ask for a dozen cans of the same article the price to you would be considerably less than if you purchased the merchandise at the retail store. Now, suppose you were to go to the very source of supply, be it the manufacturer, the farmer or the mill. Not only would there be a vast difference in price, but also a better selection in quality. It is — the power of mass buying at work.

In essence, that is what your "Group Insurance Programs" mean. We have gone direct to the manufacturer (the insurance company) and said to them that we represent many thousands of potential insurance buyers. We refuse to pay the high cost of an individual policy and furthermore, we want a broad, comprehensive and understandable policy. We want a policy that will protect the "nurse", the "anesthetist" and the "individual". We want these and expect these because we have the power of mass buying.

A good example of your "power of mass buying" is your accident and sickness, income protection plan. As an individual you cannot buy a better policy, yet through your association you have the low rates and the excellent benefits. Lifetime accident benefits have been added to the policy, at no extra premium. Up to $400 a month monthly benefits is another recent addition. These additional benefits and the ones to be added are the result of a successful, stable accident and sickness program.

The principles of group insurance are recognizable in most every market — automobile, food, clothing, building materials, travel, publication, drugs, and medicines, are a few examples. It costs the producer of the merchandise less to supply the large purchaser than to supply the individual purchaser.

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CASE REPORT

(Continued from page 57)

Company and the following paragraph is a direct quotation.

"Investigation of the concurrent use of Ilopan and succinylcholine has not been pursued. Succinylcholine is a skeletal muscle relaxant and an autonomic ganglionic blocking agent. Succinylcholine blocks the receptor sites which are normally stimulated by acetylcholine. Under prolonged surgical conditions, acetylcholine production is likely to be reduced. Since succinylcholine and acetylcholine are both split (hydrolyzed) by cholinesterase enzymes, a deficient production of acetylcholine probably would result predominantly in hydrolysis of succinylcholine. However, if acetylcholine elaboration is restored by Ilopan, the hydrolysis of succinylcholine would appear to be proportionately diminished, thus prolonging its action, but lessening the intensity of succinylcholine action."

PEGGY STEWART, C.R.N.A.
WORLEY HOSPITAL
PAMPA, TEXAS

SAN FRANCISCO

1960

ANNUAL MEETING

August 29 to September 1

Assembly of School Directors

August 28

Hotel Headquarters

SHERATON-PALACE

HALE

(Continued from page 44)

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RATTAN

(Continued from page 55)

The induction should not begin until the patient is draped and the operating team in complete readiness. The surgeon must then proceed with all due dispatch, in order to avoid serious difficulties with resuscitation of the infant.

SUMMARY

There is no "best" method for obstetrical anesthesia. There is a best method for a particular patient, obstetrician, and anesthetist, in a particular hospital at a particular time. The job of the obstetrical team is to find that method, and to use it properly. Of all the variables to be considered, the most important by far is the competence and experience of the individuals involved. A poor method in expert hands is infinitely safer than a good one which is inexpertly employed.

The THIRTY-FIRST QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on May 14, 1960. The deadline for accepting completed applications including the transcripts is April 1. Notice of eligibility will be mailed about April 10.

Hospital Safety

Harriet L. Aberg, C.R.N.A.

DRIP-DRY——

A follow up on the wearing of "drip-dry" type uniforms in areas where flammable anesthetics are to be used.

Public Law #88 — the Federal Flammable Fabrics Act went into effect in 1954. ". . . to prohibit . . . articles of wearing apparel and fabrics which are so highly flammable as to be dangerous when worn. . . ." It eliminates from the market what we used to call "torch sweaters" etc.

However, this has nothing to do with the ability of a textile to build up static electricity.

To illustrate — given a fire in a container such as a metal wastebasket, one layer of a nylon uniform placed across the top of the container will extinguish the fire. So this textile conforms to Public Law #88 perfectly —it is difficult to flame it, in fact it chars instead. But this same nylon uniform will build up large amounts of static electricity with the friction of walking and then is dangerous in an area where flammable anesthetics are being used.

It is the latter property with which we, as anesthetists, are concerned. "Drip-dry" cottons we are quite sure comply with the requirements of the Federal Flammable Fabrics Act. That is good and is important, but whether its surface resistivity is less than 5×10^{11} ohms (per unit square of the material) is an entirely different proposition. Quoting this column in the August 1959 Journal we continue to believe that "In all probability any treated cotton including the so-called "drip-drys" is not likely to be safe for wear in areas where flammable anesthetics are to be administered. We would advocate that only unmodified cotton uniforms be worn."

Question — With conductive floors, conductive furniture and casters, and conductive mattresses on the operating room tables, may we assume the patients are also grounded?

Referring to N.F.P.A. #56—Code for Use of Flammable Anesthetics, 3552 and Appendix B - 4 - (c) - "Electrical connection of the patient to the conductive floor shall be assured by a conductive strap in contact with the patient's skin with one end of the strap fastened to the metal frame of an operating table or shall be electrically interconnected by other means."

With the conductive floor as a base —persons are electrically connected to it by means of soles of feet through conductive shoes or by conductive straps from shoe soles to the legs. Furniture is connected to it by means of conductive casters or feet, and patients are connected to it through the mattress or framework of the

operating table through casters and brake feet to the floor.

All of these points of contact must be just that. Shoes must not be isolated by a film of wax, powder or dirt. Casters must not be isolated by a film of wax, powder or dirt, or suture material wound around the pin, spindle or axle of the wheel or caster. The patient must not be isolated from the mattress and table by nonconductive sheets. Because such covering over a conductive mattress can isolate the patient from the conductive floor, the Committee on Hospital Operating Rooms included in #56 the sections quoted above. This conductive strap can be of metal, conductive rubber or even some method using water's excellent conductive property.

Whether or not the patient is grounded is not too important. But it is of the utmost importance to know whether or not he is in electrical contact with a conductive floor and that all persons and objects in the room are electrically connected. As a matter of fact, most conductive floors become grounded when they are in contact with heating pipes, water pipes and sewage connections. #56—Appendix A 252—Conductive Flooring—"To be effective it is necessary only that it be conductive and that the persons and objects be electrically connected to it."

Miss Aberg is A.A.N.A.'s representative on the N.F.P.A. Committee on Hospital Operating Rooms.

Any questions pertaining to hospital safety may be directed to the Executive Office. Answers will be included in this section in future issues.

Legislation

Emanuel Hayt, LLB., Counsel A.A.N.A.

Surgeon Liable for Failure of Nurse Anesthetist and House Staff to Note Patient's Allergy to Penicillin

On September 13, 1956, plaintiff was working for the Baton Construction Company, when a nail ricocheted from a ramset gun he was using, entered his right leg and fractured the fibula. He was brought to the receiving ward of Episcopal Hospital between 2 and 3 p.m. The gist of his complaint is that thereafter he became the patient of defendant Dr. Edgar L. Pennell, Jr., who negligently permitted his agents and servants to prescribe penicillin for plaintiff although they knew or should have known that plaintiff was allergic to this drug, as a result of which plaintiff sustained serious permanent injuries. The injuries were clearly proved and there is no contention but that they were caused by the penicillin.

A few months before his accident plaintiff had contracted a virus condition. His family physician, Dr. Katzman, had given him one injection of penicillin in treating him for it. Plaintiff developed a skin rash resulting from an allergy to this drug, whereupon Dr. Katzman discontinued its use and wrote a note on one of his prescription blanks which stated that plaintiff was allergic to penicillin and that he was never to receive that drug under any circumstances.

While plaintiff was in the receiving ward he showed the note to one of the nurses and to Rex, the junior intern. Plaintiff remained in the receiving ward about four hours. During this time Dr. Pennell came there and plaintiff's wife spoke to him, complaining about the long time plaintiff was kept waiting to have the nail taken out of his leg.

While plaintiff was still in the receiving ward, Dr. Hatemi was called to the ward. Dr. Hatemi was at that time a graduate of a medical school in Iran. Dr. Hatemi was a paid employee of Episcopal Hospital. In his capacity as a resident he was duty-bound to act on behalf of the hospital. In accordance with that duty he was called upon to see plaintiff upon admission. He made an examination or partial examination of plaintiff. However, under the law, in view of the fact that he was not a licensed surgeon, he could not himself undertake operative surgery without first consulting and receiving approval of the chief or an associate of Surgical Service B. He therefore consulted with Dr. Pennell who authorized him to proceed with the operation. At this time, in accordance with the rules of the hospital and as demonstrated by Dr. Pennell's later conduct, the plaintiff became the patient of Dr. Pennell. Dr. Pennell had

the choice of operating himself or of designating another qualified person to do it for him. He chose Dr. Hatemi to operate in his stead. But when he did so he did not abandon, in contemplation of the law, either the right of control or his own interest and duty in the premises. It is clear, said the Court, that from this moment onward Dr. Hatemi was the Agent of Dr. Pennell. His negligence thereafter, if any, is in the light of the verdict imputable to Dr. Pennell. What did Dr. Hatemi do? He directed Rex, the junior intern, to take a history of the case. The evidence is clear that the general duties of Rex qualified him to take case histories. However, in the normal course of events, since the hospital had assigned him to the receiving ward, he would not have taken this history had he not been selected by Dr. Hatemi to do so. In this sense, therefore, Rex was "borrowed" from the hospital. While remaining a general employee of the hospital he became a sub-agent of Dr. Pennell. He took the history and although informed of the plaintiff's allergy to penicillin, failed to note it on the chart. The defendant's chosen agent, Dr. Hatemi, performed the operation and was therefore in full command of all persons in the operating room to the exclusion of hospital control during that period.

Rex informed the nurse anesthetist of the fact that the history was incomplete. Exactly what followed is not known. If the nurse did as Rex asked and noted the allergy on the chart before Dr. Hatemi read it, or told him of it, a jury could find that Dr. Hatemi himself was negligent. If the nurse failed to correct the history or inform Dr. Hatemi her negligence is apparent. If Dr. Pennell had been in charge of the operation in place of Dr. Hatemi would he have been responsible for disregarding the correction or for Rex's and the nurse's failure? If so, then he is equally responsible here since his chosen agent, Dr. Hatemi, was acting for him. The knowledge of Rex and the nurse anesthetist and that imputable to Dr. Hatemi was imputable to Dr. Pennell.

We conclude that the harm suffered by plaintiff was caused by the postoperative orders given by Dr. Hatemi during the surgery itself at a time when he was responsible for the acts of both Rex and the nurse anesthetist. It follows from a proper application of the principles of respondeat superior that the jury were justified in finding that Dr. Pennell was therefore responsible.

(Yorston v. Pennell, 9 CCH Neg. Cases 2d 1009-Penn.)

Abstracts

Brown, A. S.: Treatment of intractable pain by subarachnoid injection of carbolic acid. Lancet 2: 975-978 (Nov. 8) 1958.

"The intractable pain of peripheral disease is usually transmitted over a number of nerve-roots. Hence, if it is to relieve such pain, the interruption of nerve conduction by surgical or chemical means may have to be so extensive that it leaves the patient with a severe disability. . . . In 1955, Maher reported that certain intrathecal solutions of carbolic acid abolished pain completely in some patients without adding to their disabilities. . . .

"I chose 'Myodil' as the solvent, in preference to glycerin; the action of myodil in the subarachnoid space is known, whereas that of glycerin is not. I also felt that the use of glycerin and carbolic acid together would make assessment of the cause of any complication unnecessarily difficult. A patient with uncontrolled pain in the terminal stages of malignant disease was offered this treatment. . . .

"Three or four other patients with widespread malignant disease were treated similarly, the concentration of carbolic acid being increased from 1/20 to 1/15 without untoward effects. There is never any doubt about the result. Pain either persists or is abolished. . . . During 1957 fifty-five patients were treated for severe intractable pain by subarachnoid injections of carbolic acid. . . .

"The method appears to be safe . . . but a much longer follow-up period will be required to exclude the possibility of serious delayed neurological complications. Such complications may take months, or even years, to develop; but, once established, they cannot be reversed. For this reason intrathecal carbolic acid should be used mainly in treating the pain of malignant disease until we can be reasonably certain that it does not cause serious late complications."

Dyrberg, Viggo and Andersen, E. W.: Postoperative analgesia with R 875. A comparison of the effects of dextro 2,2-diphenyl-3-methyl-4-morpholino-butyrylpyrrolidine and morphine in man. Acta chir. Scandinav. 115: 243-248, 1958.

"Our immediate aim has been to test the analgesic action of the diphenylpropylamine R 875 against that of morphine in post-operative pain. . . . Two hundred patients of both sexes between the ages of 20 and 65 years who were prepared for major elective surgery, and in whom postoperative pain could be anticipated, were selected for study. . . .

"The anaesthetic procedure was standardized as follows: one and a half hours before operation each patient received a subcutaneous injection of pethidine 75 mg. and atropin 0.5 mg. Anaesthesia was induced with thiopentone and succinylcholine, orotracheal intubation was then carried out, and anaesthesia maintained with nitrous oxide and oxygen (3.5 litres to 1.5 litres) with d-tubocurarine as required. During anaesthesia nitrous oxide was the sole analgesic. . . .

"R 875 was found to be slightly more effective than morphine as an analgesic agent, when both drugs were employed in doses of 10 mg. The duration of action is similar to that of morphine. An attempt to gain an impression of the incidence of side actions proved unsuccessful, which confirms previous findings by other investigators. Recent suggestions in favor of using the result of the first postoperative dose of analgesics as the yardstick to test new analgesics against standards were taken into consideration and seem to merit further study."

Reinberger, J. R. and Mackey, W. F.: Cardiovascular reactions to pitocin. Am. J. Obst. & Gynec. 76: 288-291 (Aug.) 1958.

"The parenteral administration of posterior pituitary extracts for prevention or control of postpartum hemorrhage has been a routine procedure for almost half a century. This practice has been so successful that one now hesitates to leave the newly postpartum patient until one of the posterior pituitary extracts has been given. And yet this administration is not without danger. . . .

"The combination of two parasympathetic stimulants, cyclopropane and pituitary extract, is most dangerous. In the circulatory system, they have a synergistic tendency to produce hypertension and cardiac arrhythmias. It is important, therefore, never to use posterior pituitary extract when an oxytocic is indicated in a patient under cyclopropane anesthesia.

"A survey of hospitals reveals that more attention should be directed to the type of posterior pituitary extract being used in the departments of obstetrics. The department of obstetrics should not have any surgical Pituitrin for this preparation should never be used as an oxytocic. The department of surgical gynecology should have both obstetrical and surgical Pituitrin and, if prescribed, the type of Pituitrin should be specifically ordered; otherwise, surgical Pituitrin could be given. . . .

"We have now discontinued the use of Pitocin or any drug in the uterus at the time of cesarean section. . . . Intravenous Pitocin after normal delivery has just lately been shown to cause a more frequent and higher rise in blood pressure than either Ergotrate or Methergine. This is very important, especially since it implies that Pitocin is dangerous in toxemia."

ANESTHESIA ABSTRACTS

edited by

- John S. Lundy, M.D.
- Florence A. McQuillen, R.N.

Current literature is selected for its value to the nurse anesthetist, condensed into abstracts, published two to three times yearly in **Anesthesia Abstracts.** Request a single copy or place a "standing order" to receive each volume as published.

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Book Reviews

TEXTBOOK OF ANATOMY AND PHYSIOLOGY. By Catherine Parker Anthony, B.A., M.S., R.N., Assistant Professor of Nursing, Science Department, Frances Payne Bolton School of Nursing, Western Reserve University; formerly Instructor of Anatomy and Physiology, Lutheran Hospital, Cleveland, Ohio; formerly Instructor of Anatomy and Physiology, St. Luke's Hospital, Cleveland, Ohio; formerly Assistant Instructor of Anatomy and Physiology, Frances Payne Bolton School of Nursing, Western Reserve University, St. Louis, Mo., The C. V. Mosby Co. Cloth. 574 pages, illustrated, 5th ed., 1959. $5.35.

The first edition of this book appeared 15 years ago and was written primarily for student nurses. This edition has been brought up to date with greater emphasis on physiology.

A chapter on Fluid and Electrolyte Balance and a chapter on Acid-Base Balance have been added. The portions on cell and muscle physiology, metabolism, reproduction, the hypothalmus and the endocrines have been rewritten. Changes have been made in the review questions at the end of each chapter.

Features retained are the outline surveys at the beginning of each chapter, the outline summaries at the end of each and the many excellent illustrations. A Trans-Vision® insert of the Anatomy of the Torso is included.

A glossary and revised supplementary reading suggestions are found at the close of the text.

This book presents the basic facts of body structure and function and will be especially valuable to anesthetists who are engaged in teaching.

SURGICAL SERVICE GUIDE. By Louis T. Palumbo, M.D., M.S., F.A.C.S., Chief, Surgical Service, Veterans Administration Center, Des Moines, Iowa; Clinical Professor of Surgery, College of Medicine, State University of Iowa. Chicago Ill., The Year Book Publishers, Inc. Cloth. 208 pages, 1959. $6.00.

This guide provides a basis for the management of the usual surgical patient.

The first chapter summarizes some of the duties and responsibilities of the anesthetist. 82 pages deal with orders and postoperative management. The remaining portion is devoted to Complications, Fluids and Electrolytes, Diagnostic Procedures, basic features of pediatric surgery, certain phases of gynecology, Follow-up Procedures and Dietetics. Many good illustrations are included.

Anesthetists will find much in this guide that is useful.

CLINICAL COORDINATION OF ANATOMY AND PHYSIOLOGY. By Martha Pitel, R.N., Ph.D., University of Rochester; formerly Yale University and Mildred Schellig, M.D., State University of New York, Harpur College. New York, N. Y., Springer Publishing Company, Inc., Paper. 320 pages, 1959. $5.50.

This text is presented in workbook form. The authors relate anatomy with surgery and physiology with medicine.

Many excellent drawings are included. Questions follow each section. The bibliography lists many references.

This book will especially appeal to anesthetists involved in teaching.

Classified Advertisements

NURSE ANESTHETIST — 154 bed hospital—suburban community North of Chicago on Lake Michigan's beautiful shoreline. New surgery and other facilities recently constructed. Department of four Nurse Anesthetists under an M.D. Anesthetist. Salary open depending upon qualifications. Merit increases. Excellent living facilities. For further details write Personnel Director, Highland Park Hospital Foundation, Highland Park, Illinois.

REGISTERED NURSE ANESTHETISTS: Immediate openings for permanent employment. 670 bed hospital. Exceptional opportunity for well trained Nurse Anesthetist in active operating room suite. Apply: Personnel Director, Harper Hospital, Detroit 1, Michigan.

NURSE ANESTHETIST: 364 bed General Hospital being enlarged to 500 beds. Want to enlarge present staff of one M.D. plus 7 Anesthetists. Salary from $400 to $500 per month, plus extra bonus payment per case on call duty and retirement and sickness benefits. New air conditioned Operating Rooms. Apply Chief, Department of Anesthesia, York Hospital, York, Pa.

ANESTHETIST—330 bed voluntary general hospital—not tax supported. Modern air-conditioned surgical suite. Excellent working conditions. Room and board available if desired. Staff consists of 6 nurse anesthetists under supervision of 3 anesthesiologists. Salary open. Apply Decatur and Macon County Hospital, Decatur, Illinois.

NURSE ANESTHETIST—New and Modern Surgery: unusually strong and well diversified Surgical Staff. Good opportunity in new 260-bed expanding hospital; college town location; good personnel policies; 40-hour week; 7 holidays, hospitalization, Social Security. Apply: F. J. O'Brien, Administrator, Chambersburg Hospital, Chambersburg, Pa.

NURSE ANESTHETISTS for 220 bed community hospital. Working with private group. Two full time M.D.'s, four nurses, all agents and techniques. Modernization program going on. Two and one-half hours from Boston and New York. Write G. J. Carroll, M.D., William W. Backus Hospital, Norwich, Connecticut.

NURSE ANESTHETIST — 125 bed Genral Hospital, Southeastern Massachusetts, to work with Anesthesiologist. Salary, etc. open depending on training and experience. Write: William H. Lewis, M.D., 376 Tremont St., Taunton, Mass.

REGISTERED NURSE ANESTHETIST. Excellent working conditions in modern 132-bed hospital. Friendly community with two colleges. Beginning salary $500 plus call pay. Apply Ralph B. Bersell, Administrator, Passavant Memorial Area Hospital, Jacksonville, Illinois.

WANTED: Nurse Anesthetist for Obstetrical Anesthesia night shift or if preferred, Surgical and Obstetrical Anesthesia. Apply: R. R. Lamb, M.D., 527 Rutherford Ave., Trenton, New Jersey.

NURSE ANESTHETIST — New 50 bed hospital; excellent working conditions and Personnel Policies; located near Cincinnati; contact Administrator, Dearborn County Hospital, P. O. Box 72, Lawrenceburg, Indiana, Phone 1010.

ANESTHETIST—A.A.N.A. member, for 325 bed hospital 16 miles West of Chicago's Loop. Good salary commensurate with experience, 4 weeks vacation, paid life insurance, Blue Cross available, sick leave, paid holidays and other liberal benefits. Alternate call duty with five other Anesthetists. Emergency OB call only, with fee for OB call. Apply Personnel Director, Memorial Hospital, Elmhurst, Illinois.

OPENING for 2 Registered Nurse Anesthetists. University City, population 100,000. New 175 bed hospital, 2 M.D. Anesthesiologists and 4 R.N.A.'s now in department. Busy OB and Surgery. Day off after call. Must be energetic and willing to adopt new methods. Sick leave. 2 weeks vacation to start. Retirement plan and other benefits. Congenial working conditions. Begin $500 per month. Contact either Dr. Francis or Miss Chasteen, R.N.A., Central Baptist Hospital, Lexington, Kentucky.

NURSE ANESTHETISTS (4) — To increase present staff. Accredited 500 bed hospital in University town. Excellent salary; liberal Personnel Policies. Write Administrator, St. Joseph Mercy Hospital, Ann Arbor, Michigan.

Lutheran Deaconess Hospital, a 200 bed General Hospital located on the near Northwest side of Chicago is in need of an anesthetist for a permanent, full time position. For details write to the Executive Director, Lutheran Deaconess Hospital, 1138 N. Leavitt St., Chicago 22, Illinois.

NURSE ANESTHETISTS — for a 125 bed General Hospital, to expand to 200 beds, located in Northern Illinois. Salary $625.00 per month to start with good fringe benefits. Write or call Administrator, Deaconess Hospital, Freeport, Ill.

URGENT NEED for Nurse Anesthetists, Male or Female, 329 bed fully approved General Hospital in Concord, North Carolina. County owned, population approximately 64,000. Annual vacation, 6 holidays, accumulative sick leave, Social Security, hospitalization insurance paid by hospital. Operating Room air conditioned. All Heidbrink equipment. Department under supervision of Anesthesiologist. Covering OB and Surgery calls. Salary $525 - $600 depending on experience and quallifications. Extra pay for overtime and night call. Contact Hospital Administrator, Miss Louise Harkey, R.N., Cabarrus Memorial Hospital, Concord, N. C.

NURSE ANESTHETIST — woman preferred — for 85 bed accredited hospital. Beginning salary $450 and complete maintenance in attractive nurses residence. Four weeks vacation — 12 days sick leave and six holidays. Alternate call with one other anesthetist. Apply: Administrator, Lutheran Hospital, Beaver Dam, Wisconsin.

NURSE ANESTHETIST: for accredited 393 bed General Hospital. 40 hour week. No call. Top salary. Liberal vacation, sick leave, other benefits. Quarters, laundry and maintenance available. Write Personnel Director, Miller Hospital, St. Paul, Minn.

NURSE ANESTHETIST, fully accredited hospital, 160 beds plus 33 bassinets, 28 days paid vacation per year plus 7 paid holidays, uniforms furnished and laundered, sick leave and hospitalization. 8 miles from Pa. State University. $500 - $600 per month salary. Apply Administrator, Centre County Hospital, Bellefonte, Pa.

NURSE ANESTHETIST — Anesthesia Department comprised of 4 M.D.'s and 5 R.N.A.'s in 300 bed General Hospital. Apts. available. Starting salary $400.00 per month. Apply Mr. Currier, Elizabeth General Hospital, 925 E. Jersey St., Elizabeth, N. J.

NURSE ANESTHETIST: Immediate opening, 71 bed General Hospital in Montana College Town of 12,000. Call duty shared with another Anesthetist, no OB. Average 50 surgeries monthly. Salary $450 per month with full maintenance, $500 per month without. Write or call collect, Don Showman, Administrator, Kennedy Deaconess Hospital, Havre, Montana.

NURSE ANESTHETIST — For 120 bed Community Hospital in Western North Carolina to work with other Nurse Anesthetists. Hospital fully approved by JCAH; excellent salary; liberal Personnel Policies. Write Administrator, Grace Hospital, Inc., Box 150, Morganton, North Carolina.

WANTED — 2 NURSE ANESTHETISTS to complete staff of seven. No night call. No OB. Above average salary for area. Liberal vacation and sick leave plus choice of two pension plans. 335 bed County & Private Hospital. Apply to Personnel Manager, Duval Medical Center, 2000 Jefferson St., Jacksonville 8, Fla.

NURSE ANESTHETISTS: University Hospital. All types of Anesthesia and Surgery. Salary from $4836.00. Apply D. C. Weaver, M.D., Grace-New Haven Community Hospital, Unit of Yale-New Haven Medical Center, P. O. Box 1001, New Haven 4, Connecticut.

SURGICAL NURSE ANESTHETIST
150 bed General Hospital

●

Maximum Postings
8 daily
3 Anesthetists

●

$400 month
Full Maintenance
$80 month Apartment Allowance
Retirement Plan
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Liberal Vacation Policy

●

Pleasant community, where the Susquehanna River flows into Chesapeake Bay. Quite convenient to Washington. Baltimore, Philadelphia, New York and nearby seashore resorts.

HARFORD MEMORIAL HOSPITAL
HAVRE DE GRACE, MARYLAND

NURSE ANESTHETIST for 284 bed modern General Hospital in Michigan. Salary $5688 to $6900. For further details write Personnel Director, McLaren General Hospital, 401 Ballenger Highway, Flint 2, Michigan.

WANTED: Relief Anesthetist to take half call. Very few night calls and small OB service. Must be proficient in endotracheal technic. Ideal position for someone wishing semi-retirement. Apply Phyllis A. Roberts, C.R.N.A., Administrator, Green County Hospital, Jefferson, Iowa.

OPENING available Oct. 1960 (for Nurse Anesthetist) for 30 bed hospital in small Northeastern Oregon community. Low surgical volume and no OB allows time for personal activities or other hospital duties with additional pay. This is an ideal situation for a married woman or an older person wanting to slow down or avoid heavy work schedules. 3 weeks vacation. Salary open. Apply Administrator, WALLOWA MEMORIAL HOSPITAL, Enterprise, Oregon.

NURSE ANESTHETIST: 225 bed Children's Hospital located North Side Chicago near Lake Michigan. Starting salary $460 per month. Liberal benefits include four weeks vacation. Write: Personnel Department, Children's Memorial Hospital, 700 Fullerton Ave., Chicago 14, Ill.

NURSE ANESTHETIST — permanent — no weekends — salary open. Contact Personnel Director, St. John's Hospital, 307 S. Euclid Ave., St. Louis 10, Missouri.

NURSE ANESTHETIST—The Chicago Lying-in Hospital of the University of Chicago, night duty, 12:00 midnight to 8:00 a.m. No Call — excellent fringe benefits — salary open. For further information contact Dr. P. Ouda Olson, 5841 Maryland Ave., Chicago 37, Ill.

WANTED — Female Nurse Anesthetist — Group of 8 Physicians and 12 Nurses — Good working conditions. Contact Fresno Anesthesia Group, 1270 Wishon, Fresno 4, California.

NURSE ANESTHETIST needed early 1960 for this 500 bed modern hospital. New Surgical & Obstetrical Departments. Expanded program. Variety of work. Good working conditions. Medical Anesthesiologist in charge of the department. Starting income approximately $500 a month, exact amount dependent on training and experience. Contact Director of Anesthesia, Methodist Hospital, Peoria, Illinois. When writing, state training and experience.

NURSE ANESTHETIST: 190 bed General Hospital with new complete Operating Room Suite needs additional Surgical-Obstetrical Anesthetist. Salary $550 plus additional for rotating call. City of 40,000 on Lake Huron and St. Clair River. Apply: Administrator, Port Huron Hospital, Port Huron, Michigan.

NURSE ANESTHETIST to fill staff of four — 400 bed accredited hospital — Salary $525 — Alternate OB and Surgery call every 4th night. Contact Sister Mary Eulalia, St. Joseph Mercy Hospital, Sioux City, Ia.

ANESTHETIST

Opening for Nurse Anesthetist in hospital. Seven full time anesthetists on staff, good starting salary including salary credit for experience.

Limited night call.

Excellent insurance, vacation and sick leave benefits.

Call or write the Director of Personnel
St. Barnabas Hospital
714 - 9th Ave. South
Minneapolis, Minnesota

REFRESHER COURSE for Nurse Anesthetists; All agents, drugs and techniques used. Duration of Course from three to six months depending on need of individual. For information write Virginia L. DeMaio, C.R.N.A., Director, School of Anesthesia, The Memorial Hospital, Danville, Virginia.

NURSE ANESTHETIST. Modern Hospital, fully accredited. Expanding staff. Excellent working conditions. Starting salary $575. Apply Mrs. C. Little, Chief Anesthetist, Weirton General Hospital, Weirton, West Virginia.

NURSE ANESTHETISTS to complete staff of three for 85 adult bed hospital. Situated midway on Pennsylvania Turnpike between Pittsburgh and Harrisburg. Famous Resort Area. Salary open, liberal Personnel Policies — Apply Miss M. Valigorsky, C.R.N.A., Memorial Hospital of Bedford County or telephone Collect — Bedford 655. Route 1, Everett, Pa.

WANTED: Nurse Anesthetists for University Hospitals. Good salary, vacation and working conditions. Opportunities provided for working toward university degree. Department under direction of M.D. Anesthesiologist. Apply Robert A. Hingson, M.D., Director of Anesthesia, University Hospitals of Cleveland, 2065 Adelbert Road, Cleveland 6, Ohio.

NURSE ANESTHETIST: 240 bed hospital 60 miles East of Pittsburgh. Start $5460 - $6000 based on experience. Anesthesiologist in charge. Excellent policies including a retirement program & life insurance benefits. Contact: Personnel Director, Mercy Hospital, Johnstown, Pa.

ANESTHETIST needed by January 20, 1960. Sixty-one bed hospital with new addition in progress. Located in beautiful Shenandoah Valley and Blue Ridge Mountains, near Richmond, and Washington, D. C. Inexperienced salary start $450 per month, raise in six months. Experienced $475 and raise. Shoes and uniforms furnished. Excellent staff. Mildred E. Sydnam, C.R.N.A., Head Anesthetist, Waynesboro Community Hospital, Waynesboro, Va.

NURSE ANESTHETIST—To complete staff of 4 and 1 Anesthesiologist. 5 day week with rotating call. All agents and techniques used. Blue Cross provided — Blue Shield optional. 2 weeks paid vacation, 10 day sick leave accumulative to 30 days, 6 paid holidays, salary open. For details write to Personnel Director, St. Anthony de Padua Hospital, 2875 W. 19th St., Chicago 23, Ill.

Applications being accepted for expanding Anesthesia Department in large Catholic Hospital in Metropolitan Chicago. Liberal Personnel Policies—pension plan, credit union, automatic salary increases. Other Nurse Anesthetists employed. Reply: Box B-42, Journal American Association of Nurse Anesthetists, Prudential Plaza, Suite 3010, Chicago 1, Ill.

WANTED—Certified Nurse Anesthetist for Oral Surgery Office in Norwalk, Conn. Norwalk is on Long Island Sound. Good beaches — swimming in summer and ice skating in winter. 44 miles from New York City. Write for more details. Dr. Franklin J. Goldman, One Mott Ave., Norwalk, Conn.

NURSE ANESTHETIST—To complete staff of 4 in 500 bed University affiliated Hospital. Salary according to experience with periodic increases, range $4425 to $8330. Call taken at home. No scheduled cases on Saturdays. Additional information from Dr. Alfred M. Keirle, Chief, Surgical Service, Veterans Administration Hospital, 3200 Vine St., Cincinnati 20, Ohio.

WANTED: Additional Nurse Anesthetist for a 60 bed hospital. Some Surgical Anesthesia with Obstetrical Anesthesia. For further information write to Mrs. D. V. Martin, Administrator, Brown Memorial Hospital, Conneaut, Ohio.

WANTED: Nurse Anesthetist in a 110 bed General Hospital in beautiful Vermont town of 10,000 close to excellent ski areas. No Neuro or Heart Surgery. Approximately 1200 Surgical and 500 Obstetrical Anesthetics yearly. Department headed by Board Certified Anesthesiologist. One Nurse Anesthetist now; second needed. Excellent working conditions. Salary open. Contact F. L. Herrick, M.D., Director of Anesthesiology, Henry W. Putnam Memorial Hospital, Bennington, Vt.

NURSE ANESTHETIST — 143 bed General Hospital. Starting salary of $450.00 and complete maintenance. 5 day week with vacation, holidays, and sick leave. Contact Administrator, Bristol Memorial Hospital, Bristol, Tenn.

NURSE ANESTHETIST — 325 bed General Hospital — Surgical Service only. Excellent salary—call time paid at rate time and ½ and double time, for Sundays and Holidays. Retirement policy, hospitalization — paid vacation, sick leave. Reply: Directors Office, Highland Park General Hospital, Highland Park, Mich.

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Contributions to the Journal should be typewritten, double spaced, on one side of white typewriter paper. The title of the article and the author's name and title should appear at the top of the first page. Hospital or university affiliation, and the place and time when the paper was presented to a group should be included in a footnote on the first page.

Articles for publication in the Journal of the American Association of Nurse Anesthetists are accepted with the understanding that they have not been published or accepted for publication in other journals.

Illustrations should be glossy prints. Each illustration should carry a number and the author's name. Legends for illustrations should be typed on a separate page at the end of the manuscript. Tables should be prepared each on a separate sheet with the number and legend on the same sheet. Tables should not exceed one page under ordinary circumstances. The type size of the Journal page is 4 1/2 x 6 7/8 inches. Illustrations should be adaptable to this size or one column width, 2 1/8 inches.

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Berger, Olive L.: The Use of Respirators in the Immediate Postoperative Period. J. Am. A. Nurse Anesthetists. 27:182, Aug. 1959.

Adriani, John: The Chemistry of Anesthesia. Springfield, Ill. Charles C Thomas, 1952.

Proofs will be sent to the author prior to publication.

Manuscripts should be submitted to the Editor of the Journal of the American Association of Nurse Anesthetists, Suite 3010, 130 E. Randolph St., Chicago 1, Illinois.